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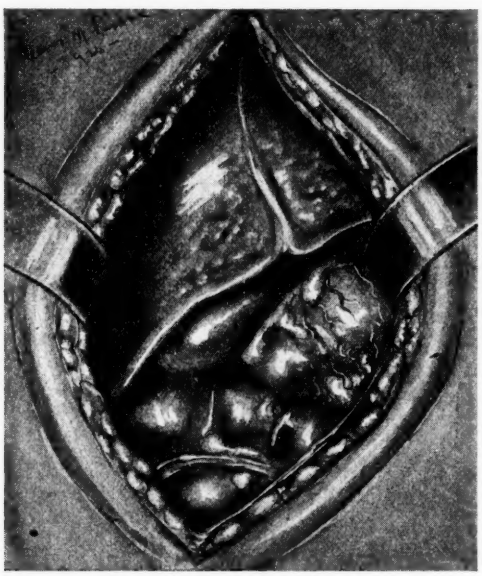
# CHOLANGITIS AND HEPATITIS*

BY JOHN B. DEAVER, M.D.†

MY apology for addressing the Bellevue Alumni Society upon cholangitis and hepatitis, still considered by many not a surgical, but a medical problem, is that my many excursions into the abdomen to undo the harm caused by biliary tract infection have revealed that with few exceptions these conditions can be remedied, or possibly permanently relieved only by mechanical interference—surgical drainage of the gall-bladder or of the common duct.

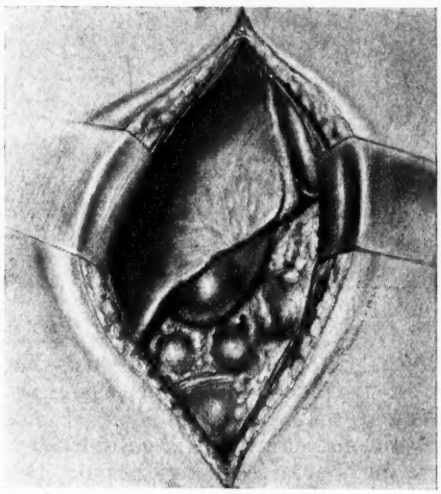


Cirrhosis of liver localized around gall-bladder region.

The pictures I show you will prove my contention. The drawings were sketched at the operating table when making the autopsy *in vivo*. Not only have I been instructed in making these dissections, but I have often been horrified to know that in this enlightened age such pathological possibilities failed to be recognized or anticipated clinically. The value of the immediate results obtained by exposing this pathology to the light of day are thus suggested anew. This is to my mind real research work, destined to bring relief to suffering humanity.

Cholangitis, blood-borne, lymphatic-borne, and duct-borne, is a clinical and pathologic entity, presenting a circumscribed, diffusing or diffuse infection of the biliary tract in which the bile channels are characteristically involved. The seriousness of the disease is dependent upon the extent of the infection, the type of the infection, the consequent derangement of liver function, and the harmful, and often permanent sequelae.

A knowledge of the minute anatomy of the bile-ducts is necessary for a complete conception of the pathology of cholangitis and hepatitis. The bile-ducts are not mere passive channels for the transit of bile, although this is their chief purpose. A close study of their structure reveals that they are lined by a modified type of columnar epithelium and that arranged along the walls of their lumen are numerous tiny evaginations. Emptying into these tiny evaginations or parietal saccules are ducts of glands which ramify most extensively in the walls of the principal bile ducts. These glands produce a mucous secretion which is collected in the parietal saccules and later is poured out into the ducts. If one will take the trouble to make histologic studies of the common bile-duct from a case of cholecystitis in which the infection is grossly confined to the gall-bladder, he will frequently find in the parietal glands of the duct evidence of inflammatory changes, as round-cell infiltration, oedema, and cystic changes. It will be apparent that these changes often accompany cholecystitis and that more extensive involve-



Localized fibrosis, circumscribed hepatitis, around gall-bladder, probably lymphogenous.

*Read before the Society of Alumni of Bellevue Hospital, New York City, December 4, 1929.

†Deaver—Surgeon-in-Chief, Lankenau Hospital, Philadelphia. For record and address of author see "This Week's Issue," page 548.

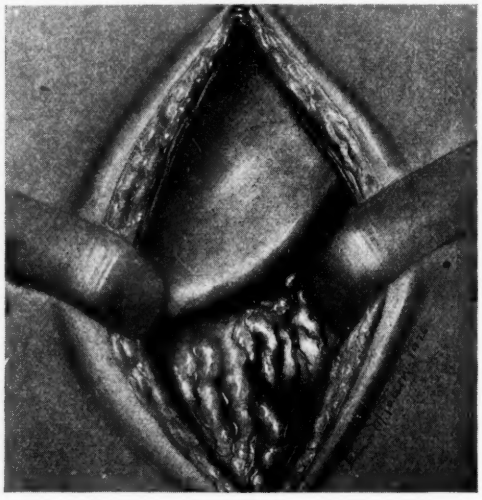
ment of the parietal glands of the ducts within and without the liver constitutes the chief pathologic feature of cholangitis. The infection in cholangitis is not confined to the surface of the lumen of the ducts, but is well entrenched deep in their walls, which accounts for the tenacity, seriousness, and for the difficult therapeutic management of this condition.

Cholangitis is usually accompanied by extension of the infection to contiguous structures. The most frequent associated lesion is cholecystitis and it is often difficult to ascertain whether the infection in the gall-bladder is the primary focus or a secondary lesion. From experimental work and clinical studies it has been demonstrated that cholecystitis may arise independently of infection in the liver. This can be accounted for by a selective localization of bacteria, or according to the doctrine of a prepared soil, the result of a primary functional derangement of the gall-bladder, or by a combination of the two. It is not difficult to conceive that many cases of cholecystitis begin as a diffuse infection of the biliary tract and when the acute condition subsides, the residual infection becomes localized in the gall-bladder, where certain factors favor its chronicity.

Thick bulging under surface of liver. Operative.

When operating for cholangitis, the surgeon frequently finds changes in the regional lymphatics, mainly enlargement of the lymph nodes in the hilus of the liver, at the root of the cystic duct, at the junction of the cystic and common ducts, along the course of the common duct and around the head of the pancreas. These nodes are often markedly swollen and may actually cause obstruction to bile-flow by pressure on the ducts. The finer lymphatic channels in the liver, especially those radiating from the region of the gall-bladder, are often distinctly seen. The condition actually constitutes a lymphangitis. The head of the pancreas is frequently swollen and soft, while the body and tail are not involved. The changes within the liver are either enlargement or congestion. Sections of the liver will show hepatitis in the form of inflammatory changes around the smaller bile-ducts, and degenerative lesions such as oedema and cloudy swelling of the parenchymal cells. The intra-hepatic lesions are usually confined to the right lobe, but in more severe cases the left lobe; the spleen may also be involved in the infectious process.

Cholangitis is sometimes encountered in the course of the infectious fevers, such as influenza, pneumonia, typhoid fever, and acute rheumatic fever. In such instances the symptoms pointing to involvement of the biliary tract are slight jaundice, tenderness, and sometimes enlargement of the liver; pain is only occasionally present. This type of cholangitis may be either toxic or infectious in origin; the jaundice is due to functional deficiency of the liver. There is also a primary type of cholangitis, infectious in origin, which begins with fever often septic in type, malaise, gastro-intestinal disturbance, and jaundice. This condition is sometimes confused with acute catarrhal jaundice, acute yellow atrophy of the liver, and mild subacute pancreatitis. In cholangitis the infectious features predominate and the jaundice is only moderate in degree. Most of these cases subside in the course of several weeks and may completely clear up. It is not unusual, however, for the condition to recur. When the symptoms do not subside in due time, or if they become aggravated, there is urgent indication for considering surgery and external biliary drainage.

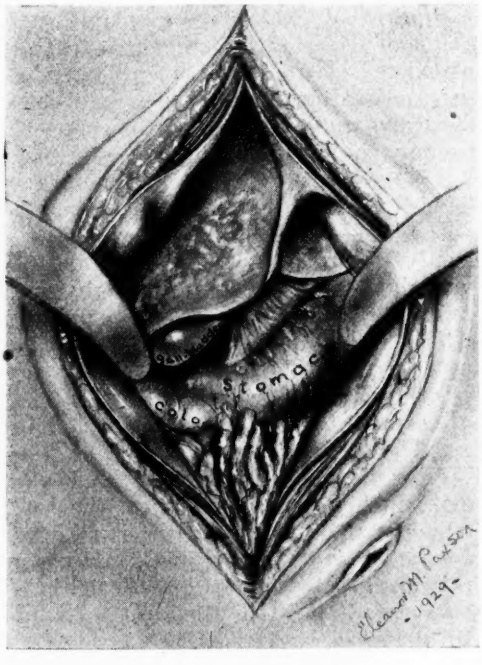


Chronic hepatitis. Rounded edge of liver, right lobe.

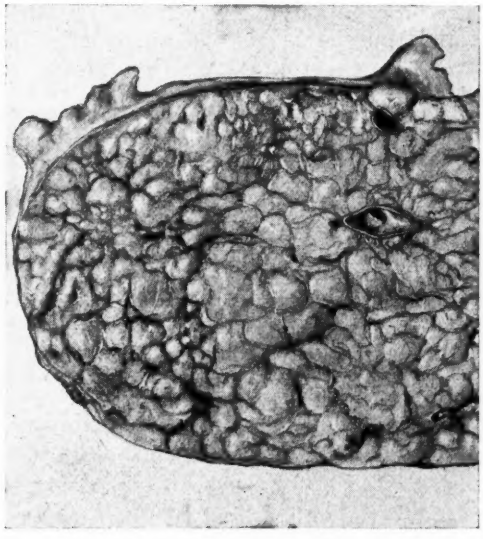
Cholangitis is also seen where the early history is one of gastro-enteritis, where the likeli-

hood of an ascending infection, either by way of the lumen of the common duct or along the neighboring lymphatics, must be considered.

Surgical experience, however, shows that in the majority of cases of cholangitis there has been some preëxisting lesion of the biliary tract with a recent acute exacerbation of infection or interference to the flow of bile. As I have previously stated, the most common associated lesion is cholecystitis and its complication, stones in the gall-bladder and in the common duct. Stone in the common duct is usually associated with a choledochitis, a deep interstitial infection. Acute exacerbation and extension of this smouldering infection in the duct ensue when there is prolonged partial or complete obstruction to bile flow. The infection may then rapidly involve the entire biliary tract, giving rise to sepsis with intermittent chills and fever, and persistent jaundice, which may partially fade only to become deeper as the obstruction increases. If intervention is too long delayed, the condition goes on to the formation of pus within the ducts as well as in the gall-bladder, and of multiple large or small abscesses within the liver itself. The gravity of the situation becomes proportionally greater as the surgeon's help is withheld, until the vital function of the liver has been destroyed beyond hope of redemption by any means whatsoever. Under such circumstances the liver does not regenerate, as after removal of a portion of it. Stricture of the common duct may closely simulate calculous obstruction because the symptoms of both, with the exception of pain, are essentially those of cholangitis. A stricture may cause only partial obstruction to the bile flow, but with insidious, progressive, and often symptomless harmful effects on the liver, the result of back pressure. This condition is analogous to the renal damage due to urethral stricture and to chronic prostatic obstruction. Acute obstruction to bile flow occurs when there is reactivation of the infection about the stricture, with oedema and occlusion of the lumen. In the case of the common duct, this means the onset of cholangitis.




Liver with mottled surface.



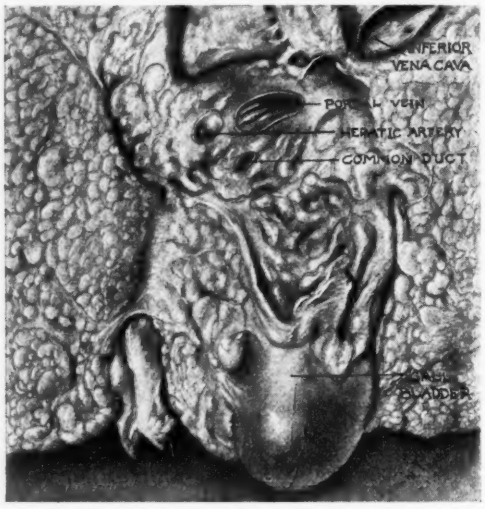
Cross section showing marked cirrhosis of liver.

Cholangitis is seen as an acute and as a chronic condition, the latter being the more troublesome; it resembles hepatitis and biliary cirrhosis and is characterized by recurrent acute attacks of fever, slight jaundice, and painful swelling of the liver.

Acute cholangitis is seen either as a mild or as a severe suppurative form; the mild type is present in the so-called catarrhal jaundice and is due to extension of infection to the liver by way of the papilla of Vater and the common duct. Gastro-duodenal irritation, the forerunner of this type of jaundice, may result in inflammation of the duodenum, which, if it involves the site of the papilla of Vater, will cause swelling of the mucous membrane around the papilla due to inflammatory oedema and partial or complete occlusion of the opening, with back pressure of bile and consequent painless jaundice. I have seen chronic jaundice and chronic cholangitis, follow catarrhal jaundice, that required drainage of the common duct to clear up the situation. Therefore, catarrhal jaundice must not always be regarded as a trifling condition. The fact that it is painless adds to its importance; we all know the significance of painless jaundice from the standpoint of diagnosis, prognosis, and treatment.

The symptoms of cholangitis are those of infection plus certain peculiar features related to the liver and its functions. In mild cases there is fever, malaise, anorexia, slight jaundice and

often an appreciable enlargement of the liver. The symptoms may persist for several weeks and the condition then subside spontaneously. An accurate diagnosis may be difficult because of confusion with catarrhal jaundice, stone in the common duct, hepatitis, and pancreatitis. Laboratory tests are helpful to a certain extent but should not be implicitly relied upon. The van den Bergh test will serve to differentiate obstructive from functional jaundice. Estimation of the amount of serum bilirubin permits a daily observation of the degree of jaundice, so that the surgeon is able to know more or less accurately the progress of this phase of the condition. However, with the aid of, or in spite




Marked cirrhosis of liver—inferior surface—region around gall-bladder (diabetic case).

of favorable laboratory reports, the surgeon must exercise his own ripe judgment based on experience and on his estimate of the patient's condition, as to the exact time for, and the extent of, the procedure to be adopted. In the severe type of cholangitis, characterized by septic fever, deep jaundice, profuse sweats, anorexia, nausea, vomiting, loss of flesh, extreme prostration and marked enlargement of the liver, the outlook is grave and while some form of drainage operation is urgently indicated, the surgeon must exercise extreme caution as to the proper time for intervention and his efforts should be confined only to the necessary minimum. In other words, the margin of safety must not be overstepped.

Certain local conditions dispose to suppurative cholangitis because they diminish the resistance of the ducts and render infection more easy. Such conditions are bile stagnation, old inflammation, gall-stones, tumors, rupture of hydatid cysts into the ducts, as well as malignant tumor of the common duct, most often at its terminal end of the duct and at the liver.

The possible complications add to the seriousness of the condition. In addition to local or general peritonitis, from leakage or rupture of the suppurating areas, there may be a general hemic infection; pus and micro-organisms may pass into the hepatic veins and so reach the lungs and give rise to pyaemic abscesses, pleurisy, and empyema; or bacterial infection may enter the general circulation and involve the joints and meninges and even the heart in the form of malignant endocarditis.

While prognosis depends on the course of the disease, treatment is essentially surgical. If the condition remains limited to the large ducts or is operated on early, before the liver or pancreas has become affected, recovery may occur, but hepatic involvement darkens the prognosis. If multiple abscess formation and diffuse suppuration of the portal spaces take place, death is inevitable, but where there is only a single local area of suppuration in the liver, the outlook is brighter. In addition to the course and complications of the disease, much depends on early operation and free drainage. Surgical drainage, to accomplish the most, must be made early and must be more or less prolonged—not only for weeks but for months.

The most common variety of acute cholangitis is that seen in connection with common duct stone, readily recognized and corrected by removal of the stone or stones and drainage for a short time. The severity and persistence of acute non-suppurative cholangitis depend upon the character of the infection and the length of time it has existed. This is true of all types of cholangitis, no matter what the origin. Cholangitis is not always due to disease of the common duct. It so happens, however, that three of the worst cases I have seen have followed stones in the gall-bladder, in one of which the common duct had been twice drained, although for a short time only, followed by T-tube drainage of the common duct for two years before the condition cleared up. In the second case (now eight years since the gall-bladder was removed for calculous cholecystitis), T-tube drainage of the common duct for recurrent cholangitis had been made five times at intervals of one and a half to two years. The patient is now well; but the question is for how long? In the third case, the common duct was drained twice, the last time about one year ago; the patient is still wearing the T-tube but is apparently well and has gained several pounds in weight; there is neither jaundice nor enlargement of the liver. In this last patient recovery was so slow that I thought daily treatments of diathermy over the liver worth a trial. This proved of great benefit, so much so that I now suggest it in similar cases that fail to show improvement soon after the institution of drainage. Diathermy increases the temperature of the liver, by increasing the blood supply, and also favors resolution of the periductal inflammatory exudate. Incidentally, I may say I use diathermy over the liver in all severe liver operations and with gratifying results. The good results of the use of diathermy at operation sug-

gested its post-operative use in the drained cases of cholangitis. Diathermy increases the pulse rate and produces free perspiration. Upon its discontinuance the pulse rate drops to normal and the perspiration ceases.

Medical drainage by the duodenal tube cannot accomplish the same as direct drainage, if for no other reason than that the drainage must be continuous and over a considerable length of time. This cannot be done with the duodenal tube.

Furthermore, the bacteriological findings of the duodenal washings are not verified by operative findings. For example, direct aspiration of the gall-bladder and of the common duct seldom shows micro-organisms; bacteriologic examination of the bile in the excised gall-bladder and of the bile drained from the common duct obtained by incision of the duct shows organisms in only a very small percentage of cases. This discrepancy can only be interpreted as contamination. The duodenal tube in its passage picks up organisms from the mouth and pharynx and when magnesium sulphate or some other solution is injected, there occurs both downward and reversed peristalsis; during the latter, organisms are washed back into the duodenum. When observing the escape of bile from the papilla of Vater in the open duodenum in the dog, there will be seen reversed peristalsis from the terminal duodenum and the jejunum, occurring irrespective of any irritation of the mucous membrane of the duodenum. Thus the findings are probably not those of the aspirated bile alone. This accounts for the difference in the findings of direct and indirect aspiration. I always have, and still regard the reports of duodenal washings as questionable. I also believe that by medical biliary drainage the pancreas may be subjected to the risk of organisms being carried into the duct of Wirsung, so it may be a more or less dangerous procedure. I shall be glad to hear from any of the members of this Society who practice medical drainage in biliary tract infection.

Chronic infective or catarrhal cholangitis is usually associated with, and largely due to, cholelithiasis, although it may be due to other causes. Non-calculous chronic catarrhal cholangitis may be either of the extrahepatic or intrahepatic bile ducts. The former may result from chronic gastroduodenal catarrh, as seen in alcoholics; it may occur in the course of malignant disease of the liver or of the bile ducts, especially when the growth is at the papilla of Vater; or it may result from suppuration in the liver or the rupture of hydatid cysts into the bile ducts. In such instances the cholangitis may be catarrhal at first, but is likely to become suppurative. Chronic cholangitis closely resembles biliary cirrhosis, but since the treatment for both is essentially the same, the distinction between the two is of little practical importance. In cholangitis there are recurrent attacks of fever, jaundice, and enlargement of the liver, which on direct inspection presents a mottled appearance and rounded edges; in biliary cirrhosis the liver is likewise enlarged but has a grayish or blue appearance and frequently presents radiating lines, the result of deposits of fibrous tissues.

Chronic cholangitis that does not yield to medical treatment in a reasonable time should be operated, by making T-tube drainage of the common duct. A necessary precaution is to be sure that the common and hepatic ducts are clear of stones, muddy bile or stricture and that the papilla of Vater is patulous and in case it is not it should be dilated by a series of graduated probes.

We do not know much about non-calculous chronic catarrhal cholangitis of the intrahepatic bile ducts. Poisons or bacteria conveyed to the liver either by the portal vein or the hepatic artery may give rise to long-continued inflammation of the intrahepatic ducts, or it is possible for chronic cholangitis to complicate disease of the liver, such as portal cirrhosis, chronic venous engorgement, hepatitis, and malignant growths.

In the course of this discussion hepatitis has been mentioned often enough to indicate its rôle as an accompaniment or sequel of cholangitis. It denotes inflammation of the parenchyma of the liver. Its association with cholecystitis is almost constant. At the present time it is this association and its etiological relationship to cholecystitis that lend it interest. Marked chronic hepatitis may exist with only slight changes in the gall-bladder, while even in advanced destructive cholecystitis the liver grossly may appear normal. The consensus of opinion at this time seems to be that when there is visual evidence of hepatitis, the gall-bladder should be removed. This brings up the question, will removal under these conditions be as effective as if drainage had been substituted for removal?

The most important factor in the treatment of cholangitis is its surgical management. Regardless of its type, an early surgical opinion should be sought and the case should preferably be directly under the surgeon's care. Any form of temporizing or medical treatment without surgical guidance is usually hazardous. This applies particularly to the primary type of cholangitis. The early surgical treatment of lesions of the biliary tract should be more widely adopted, especially since present-day diagnostic methods, as cholecystography, blood chemistry studies, the van den Bergh and icterus index tests, and other liver functional tests, have increased the percentage of early diagnoses. An appeal for the surgeon's help early in the course of the disease will do much to lower the mortality of disease of the biliary tract, to curtail morbidity figures, and to reduce the menace of such complications and sequelae as cholangitis, chronic pancreatitis, hepatitis and biliary cirrhosis. The actual surgical management of cholangitis requires careful and mature judgment. Drainage

is the chief objective of any procedure that may be adopted. The manner in which this is to be attained must be determined by the conditions found at operation. The best immediate interests of the patient are of first importance, rather than ambitious surgical procedures. When the condition is acute, external drainage of bile should be provided for by the most direct means. At times, this may be most easily accomplished by cholecystostomy. When there is any doubt as to the efficacy of this method, it should be supplemented by drainage of the common duct. For this purpose it is our practice to use a T-tube. By the use of this method of drainage it is possible to maintain an external escape for the bile over as long a period as desired. The use of the T-tube serves several purposes: it provides for the external drainage of bile with the escape of infection; it reduces intraductal and intrahepatic tension, and also permits the bile to flow down the common duct and into the duodenum. Removal of the T-tube is accomplished by slow, continuous, gentle traction until it pops out; and is without injurious effects. We very seldom use internal biliary drainage by cholecystogastrostomy or cholecystoduodenostomy, not only because in cases of infection external biliary drainage is preferable, but because the stoma of such an anastomosis does not long remain patent if the common duct is not permanently obstructed, and the presence of an anastomotic opening may actually favor the ascent of infection from the stomach or duodenum into the gall-bladder and the upper biliary tract.

## EPIDEMIC MENINGITIS CONTROL*

BY GAYLORD W. ANDERSON, M.D.,† AND GEORGE H. BIGELOW, M.D.†

THE past year has seen a definite increase in the incidence of epidemic cerebro-spinal meningitis, affecting all parts of the country. It is true that for years we have had sporadic cases, occurring without apparent relationship to one another and due, we have assumed, to infection through unrecognized carriers. Beginning in 1926 there was a perceptible increase in incidence in a number of cities in the Middle West leading to an increased prevalence in 1927. In 1928 the incidence of meningitis in the country as a whole was higher than in any year since 1921. 1929 saw a definite outbreak on the Pacific coast, where a virulent form appeared; Detroit likewise found itself faced with a real outbreak.

Although Massachusetts had more cases in 1929 than any year since 1920, we had no marked localization. It is true that a fairly large proportion of our 167 cases appeared in the Connecticut Valley, particularly the early part of the year, but it is equally true that the Worcester District had its share. The eastern portion of the State, though having fewer cases considering the concentration of population, had, however, more than a "normal" prevalence. In view of all these facts it is well to anticipate and be ready for an increased prevalence in this State in 1930.

Epidemic meningitis (known also as cerebrospinal fever, meningococcus meningitis, "spotted fever"), shows a definite seasonal prevalence coincident with cold weather and the crowding together of people in living quarters. The detailed analysis of the seasonal incidence has varied according to regions studied. A review of the cases in this State during the past thirteen years shows that the late winter and early spring are the seasons of greatest incidence, but that the actual variation throughout the year is not great. (Table I.)

TABLE I

DISTRIBUTION OF CASES BY MONTHS

| | Mass. 1916-1928 | | N. Y. C. 1910-1925 | Ohio 1913-1928[1] | |
|---|---|---|---|---|---|
| January | 9.2% | | | 7.4% | |
| February | 8.4% | 28.1 | 34.6% | 9.0% | 30.8 |
| March | 10.5% | | | 14.4% | |
| April | 11.3% | | | 15.4% | |
| May | 9.0% | 30.2 | 33.5% | 10.8% | 34.2 |
| June | 9.9% | | | 8.0% | |
| July | 7.1% | | | 6.4% | |
| August | 6.1% | 20.7 | 13.9% | 6.4% | 18.1 |
| September | 7.5% | | | 5.3% | |
| October | 7.1% | | | 5.8% | |
| November | 6.7% | 20.9 | 18% | 5.3% | 16.9 |
| December | 7.1% | | | 5.8% | |

The figures of Neal,[2] based on a somewhat larger experience in New York City, show the same seasonal differences, but to a higher degree, possibly due to greater crowding. The experience of Detroit shows that the present wave is no exception in this regard. If we are in reality on a rising tide of meningitis, then we may well expect to find that the next six months will see a marked increase of cases.

*Incidence*—The statement is frequently made that epidemic meningitis is a disease of children and of soldiers. This has been well borne out in this State, where we find that 60% of the cases are below the age of ten, and 16% are in the first year of life. (Table II.)

Neal's figures differ from ours only in a much higher proportion in the first year of life, a fact not surprising perhaps in that her series included only those under personal observation. She made a special study of the group under three months of age[3], showing that the symptoms

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at this period of life were quite different from those seen in adults or older children. The presenting symptoms often being gastro-intestinal accompanied by convulsions, it is little wonder that many of them would have been missed had not lumbar punctures been performed. We can only wonder, therefore, how many of the 49 deaths in the first year of life reported for 1928 in this State as due to convulsions, and how many of the 117 due to meningitis, not further

TABLE II

DISTRIBUTION OF CASES BY AGE GROUPS

| | Mass. 1919-1928 | N. Y. C. 1910-1925 |
|---|---|---|
| Less than 1 yr. | 16% | 25% |
| 1 - 4 | 27% | 30% |
| 5 - 9 | 16% | 16% |
| 10 - 14 | 9% } | 15% |
| 15 - 19 | 7% } | |
| 20 - 29 | 8% | 8% |
| 30 - 39 | 3% | 3% |
| 40 - 49 | 4% | 2% |
| 50 + | 3% | 0.2% |
| Unknown | 7% | — |

defined, were in reality cases of meningococcus meningitis. During an epidemic the age distribution may be quite different with a greater proportion in the older age groups, particularly early adult life. No age is immune.

Another outstanding factor in the epidemiology of the disease is the association with crowded living quarters. This was particularly noticeable during the camp outbreaks, and was equally striking in the experience of several Pacific coast ships this past spring. We might therefore expect to find a higher incidence of cases, and therefore of carriers as well, among populations living under excessively crowded conditions. Segregation in sleeping quarters of those with respiratory infection is, therefore, recommended so far as practicable.

*Organism*—All cases of epidemic meningitis are due to the meningococcus, but all meningococci are not alike. By serological reactions a large number of strains have been demonstrated, classification of which has been very unsatisfactory. The serum produced by the State Department of Public Health, as well as the anti-meningococcic sera produced by the various commercial houses and other states, is polyvalent, since it is made by the immunization of horses with many strains of the organism. Unless the organism from which a patient is suffering is included in the group to which the horse is immunized the maximum therapeutic effect will not be obtained from this serum. The practitioner should remember that do what we may to produce a serum which will cover the maximum number of strains, there will be cases encountered in which the serum that he is using, owing to this lack of specificity, does not achieve the desired results. Experience has repeatedly shown that in such cases the use of a different serum, more specific for the infecting organism, often produces striking clinical improvement.

*Quarantine*—To the best of our knowledge meningitis is a disease in the transmission of which the carrier plays the principal rôle. It is true that occasionally we encounter isolated instances in which direct contact has apparently resulted in infection, yet such instances are rare. as are also multiple cases in the same household[4]. The experience of many of our hospitals shows that meningitis patients can be cared for in open wards with no further precautions than those implied by the term "aseptic nursing technique". We may, therefore, look upon the case as mainly a therapeutic problem, the same attitude that we observe today toward poliomyelitis. But simply because we feel that a clinical case is only rarely the source of secondary infection is not an excuse for neglecting isolation. The Department of Public Health recommends that home patients be isolated and hospital patients be kept on contact precautions for at least two weeks and thereafter until all acute symptoms have disappeared.

*Contacts*—Contact with a case frequently results in the development of the carrier condition, the organisms being found in the nasopharynx. Norton[5] has recently found that the incidence of carriers among family contacts may be as high as 65%, but that in the majority of these the duration of the carrier condition is relatively short. Routine culturing of all contacts to detect carriers has been tried and shown not to be practicable, yielding inaccurate and incomplete information. It is, therefore, not recommended. We are, however, justified in looking upon all intimate contacts as potential sources of danger, not perhaps immediately but at least as foci for the further development of carriers. The Department of Public Health therefore recommends the quarantine of family contacts for ten days and longer if diseased conditions of the nose or throat exist.

*Diagnosis*—Every suspected case of meningitis should have a lumbar puncture at once. However strong the clinical evidence may be, accurate diagnosis is impossible without it. Particularly is this true in infants and in the early stages of the disease in adults before the classical signs of severe meningeal irritation are developed. In most, if not all, cases of meningitis a stage of septicemia occurs before localization of the infection in the meninges. Though there may be premonitory symptoms such as headache, pains in the back and loss of appetite, the onset is usually sudden with severe headache, chills and vomiting. The temperature is elevated, irregular and variable, often not as high as seen in most acute infections. The headache, which may be the most dominant of the sensory symptoms persists chiefly in the occiput, the pain extending

into the neck and back. Hyperesthesia may occur. Rigidity of the neck and spinal irritation as evidenced by Kernig's sign are almost constant findings. In severe or late cases orthotonos and opisthotonos may be found. Convulsions are not common except in small children, but tremor, strabismus and nystagmus may be observed. As meningeal irritation becomes marked signs of increased intracranial pressure may be observed, especially bulging of the fontanels in infants. The eruption is variable, often absent It may be a feature of the early septicemia. At this stage blood cultures may be positive. Many variations from the above typical picture occur, especially in infants where the symptoms are often more suggestive of gastro-intestinal disturbance.* In the typical case the spinal fluid is under increased pressure and is turbid, containing many white blood cells in which may be found gram negative diplococci, the meningococci. The same organism is often found outside of the white blood corpuscles.

If on lumbar puncture the fluid is turbid, serum should be administered immediately. Occasionally a turbid fluid means a meningitis due to other organisms than the meningococcus, but these are exceptions, especially during an epidemic. In such cases the anti-meningococcus serum will have done no harm, and if on examination the spinal fluid is shown to contain meningococci several hours will have been gained at a time when every hour counts. Administration of serum at the time of the initial puncture is, therefore, strongly recommended if the fluid is found to be turbid. The treatment of a case of meningitis is a matter to be decided entirely by the attending physician. The Department of Public Health would merely like to emphasize the importance of intensive serum treatment, extending over many days.

A study now being made of meningitis in this State shows the value and importance of early diagnosis and adequate serum treatment, particularly in children. That many days may often elapse between the onset of symptoms and the calling of a physician is due to the public's lack of appreciation of the importance of early treatment, but that often many additional days elapse before lumbar puncture is done shows that many physicians tend to delay until the meningeal symptoms are well developed, a delay which we know is often fatal. The much higher incidence of recovery among those diagnosed early and treated intensively speaks strongly for early lumbar puncture, particularly at a time when meningitis is known to be prevalent. That nine per cent. of the reported cases on which we have records have not had the aid of lumbar puncture and that in 20% of those in which it was done over three days had elapsed after the physician had first seen the case, bespeaks the hesitancy with which many practitioners undertake this operation.

*Service*—The State Department of Public Health now offers to the physicians of the State, on request, diagnostic service on a similar basis to that already available for poliomyelitis. Through such a service, conference will be held with the attending physician and if the procedure is thought advisable lumbar puncture performed. If indicated, facilities will be available for the immediate administration of the initial dose of serum. This is purely a diagnostic service. Management of the case will remain the responsibility of the attending physician.

This service has been organized and will be conducted under the direction of the Advisory Committee on epidemic meningitis to the Department, made up as follows: Dr. W. L. Aycock, Dr. James B. Ayer, Dr. Charles F. McKhann, Dr. Wilson G. Smillie, Dr. Henry R. Viets, and Dr. Hans Zinsser.

*See article by McKhann in this number of the *Journal*.

### CONCLUSIONS

1.)—Cases of meningococcus meningitis should be isolated by local boards of health for two weeks and until all acute symptoms have cleared up.
2.) Intimate contacts should be quarantined by local boards of health for ten days from their last contact with a case.
3.) Culturing to detect carriers is not recommended.
4.) The Department of Public Health offers a diagnostic service for this disease, similar to that offered in co-öperation with the Harvard Infantile Paralysis Commission.

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## MENINGOCOCCUS MENINGITIS IN INFANTS*

BY CHARLES F. MC KHANN, M.D.†

THE introduction in 1907 of a specific serum for the treatment of meningococcus meningitis made possible the separation from the highly fatal types of purulent meningitis one form in which recovery might be reasonably expected[1]. Prior to the development of the serum treatment the mortality in outbreaks of epidemic meningitis had varied from 42% to 90%. Although a small percentage of patients recovered completely, improvement was always a slow process and occurred only after a more or less prolonged period of illness. Patients who did

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not die in the early stage passed into a chronic form of the disease which frequently was characterized by the development of hydrocephalus, blindness, deafness, spastic paralysis, tremors and mental deterioration. The final outcome was fatal often after months of illness. In infants the mortality was highest, ranging between 85% and 100%, and complete recovery in patients of this age almost never occurred[2].

The situation was changed materially by the development of the specific serum therapy. Rapid, critical subsidence of symptoms occurred in many patients following only one or two intraspinal injections of the Flexner serum, and complete recovery was obtained even in very young children[3].

Following the first demonstrations of the effectiveness of the serum, individual physicians in certain centers were appointed to act as distributors of the serum, to confirm the diagnosis in suspected cases of epidemic meningitis and to aid in the technique of the treatment. In Boston, the distributor of the serum saw and treated between 1908 and 1911 sixty cases of meningococcus meningitis, with a case fatality rate of 25%[4]. Ten of these sixty cases were in infants under one year of age.

In contrast to the results obtained in this early series of cases, the fatality rate in a series of seventy-four cases treated in the Infants' and Children's Hospital of Boston, in a six-year period ending November 1, 1929, was 43%. It is our purpose to analyze this latter series to ascertain the factors operating to produce this high mortality.

The distribution of the cases into three age groups with the number of deaths, and case fatality rate in each group, is shown in Table I:

TABLE I

MENINGOCOCCUS MENINGITIS

Infants' and Children's Hospital
Sept. 1, 1923—Nov. 1, 1929
Case fatality by age groups.

| Age | Cases | Deaths | Case Fatality |
|---|---|---|---|
| Under 1 yr. | 39 | 25 | 64+% |
| 1-2 yrs. | 10 | 3 | 30 |
| 2-12 yrs. | 25 | 6 | 24 |
| | 74 | 34 | 45+% |

Fifty-two per cent. of the patients in this series were infants under one year of age. In this age group the mortality was 64% as compared with 30% and 24% in the other two groups. It is apparent that the high total mortality was due largely to the unfavorable results obtained in the treatment of the disease in infants.

The mortality figures are based upon all deaths due to the infection either in the acute stage or in the stages of complications and sequelae. Thus death from hydrocephalus several months after the meningococcus infection has subsided is included as primarily the result of and due to meningococcus infection.

The relationship of the case fatality to the duration of the disease prior to the institution of treatment is shown in Table II. Because of the variation of mortality with age, the results for the three age groups are given separately.

TABLE II

CASE FATALITY BY AGE GROUPS, IN RELATION TO DURATION OF THE DISEASE BEFORE TREATMENT

| Age Group | Duration of disease before treatment | Cases | Deaths | |
|---|---|---|---|---|
| Under 1 yr. | Under 1 wk. | 16 | 4 | |
| | 1-2 wks. | 4 | 3 | |
| | 2-4 wks. | 10 | 9 | |
| | 4-8 wks. | 7 | 7 | |
| | Over 8 wks. | 2 | 2 | |
| 1-2 yrs. | Under 1 wk. | 4 | 1 | (fulminating) |
| | 1-2 wks | 0 | 0 | |
| | 2-4 wks. | 2 | 0 | |
| | 4-8 wks. | 3 | 1 | |
| | Over 8 wks. | 1 | 1 | |
| 2-12 yrs. | Under 1 wk. | 19 | 4 | (fulminating) |
| | 1-2 wks. | 1 | 0 | |
| | 2-4 wks. | 4 | 2 | |
| | Over 4 wks. | 1 | 0 | |

The results in the cases treated within the first week of the disease show almost the same percentage of recoveries in all three age groups. It appears that fulminating cases account for many of the deaths in children over one year of age and that a prolonged duration of the disease before treatment does not preclude a favorable outcome in older children. In infants, however, the mortality rises rapidly with increasing duration prior to treatment, and the outcome is uniformly fatal in prolonged cases.

In view of the high mortality in infants not treated until late in the disease, it is disturbing to find that of the patients under 1 year of age 19 or 48 per cent. had been sick over two weeks, and 9 or 23 per cent. had been sick over four weeks before the diagnosis was made and serum treatment begun. Of the 23 patients who had been sick more than one week prior to the institution of treatment only two recovered.

Obviously if the dismal outlook for infants with meningococcus meningitis is to be brightened, there is necessary earlier diagnosis of the disease in patients of this age group, or improvement in the methods of treatment of late cases.

The diagnosis of meningococcus meningitis in infants is rendered difficult by the bizarre nature of the clinical manifestations[5, 6]. The fulminating type of the disease is uncommon, the presence of petechiae over the body is rare, and the symptoms and signs are seldom those of the classical picture of meningeal involvement. In this series of cases the symptoms were frequently referable to the gastro-intestinal tract. The infant

was taken ill with fever and irritability associated with vomiting, and at times accompanied by diarrhoea. Occasionally convulsions occurred and although there was usually detectable rigidity of the neck and back, these signs were difficult to evaluate in an irritable baby. The Kernig sign was frequently absent. If the fontanel was open it was usually tense or bulging. In the early cases the diagnosis often had to be made by exclusion. Unexplained fever with irritability, bulging fontanel (when the infant was not crying), and variable gastrointestinal symptoms seemed to warrant a diagnostic lumbar puncture. The patients in whom the diagnosis was not made early in the disease developed, after ten days or two weeks, symptoms and signs which directed the attention to the central nervous system. These manifestations were early hydrocephalus, strabismus, severe convulsions, markedly retracted neck and rigid back.

The early and late signs which served to indicate meningeal involvement seldom gave indication as to the type of infecting organism. The differentiation of meningococcus meningitis from other forms of purulent meningitis was made only by bacteriological examination of the spinal fluid. Because of the time required for accurate bacteriological diagnosis, serum was administered at once to all infants who showed on lumbar puncture a turbid spinal fluid. Subsequent treatments were not given if the infecting organism was found to be other than the meningococcus.

Antimeningococcus serum is an antibacterial serum and is effective only when brought directly into contact with the organisms. The septicemic form of the disease is rare in infants and intravenous injection of serum seldom is required. Reactions frequently accompany the intravenous injections but are unusual after intraspinal treatment[7, 8].

For treatment of the meningeal infection serum was administered intraspinally. In the effort to bring the serum into contact with the organisms, in considerable concentration, it has been our procedure to give intraspinal treatments at twelve-hour intervals for three injections, and at twenty-four-hour intervals thereafter for a variable number of treatments, depending on the response of the infective process, as evidenced by the diminution of the number of cells in the spinal fluid, the disappearance of organisms from smears and cultures, and clinical improvement in the general condition of the patient. Ordinarily two injections were made after the organisms no longer could be found in smears or cultures of the spinal fluid. One injection, usually the second, was made through the anterior fontanel into the ventricle and frequently the administration of serum was carried out alternately into the lumbar subarachnoid space, and into the ventricles. The dosage of serum in infants seldom exceeded 10 c.c. and never was larger than the amount of spinal fluid removed.

Despite this intensive treatment, occasionally with the addition of the cisterna magna as a locus of injection, most unsatisfactory results were obtained in the late cases. Progressive hydrocephalus developed in some of the patients and ultimately caused death; however, in the majority a low grade infection persisted which was resistant to therapy for periods of weeks or months, and which was found at necropsy to have remained either as a walled off abscess in one of the ventricles, or as a circumscribed area of meningitis over the cerebral cortex.

The serum used in this series of cases was the polyvalent serum prepared by the Massachusetts Department of Health. In all cases the organism obtained from the culture of the spinal fluid was tested for agglutination by the specific serum. Occasionally a lot of serum was found to be relatively ineffective against a particular strain of the organism; various brands of serum, and different lots of the same brand then were tested against the organism, and for subsequent treatments the lot or brand of serum was used which showed the highest potency against the particular strain.

*Case*: An infant of six months was taken acutely ill with vomiting and fever, followed in two days by convulsions. On physical examination the infant was found to be hyperirritable; the anterior fontanel bulged prominently and was quite tense; there were no other signs of meningitis. Spinal fluid examination confirmed the impression of meningitis and demonstrated the meningococcus to be the causative agent. The patient failed to show any response to several injections of one lot of serum; the organisms persisted in smear and in culture and the spinal fluid cell count did not diminish. Another lot of the same brand of serum was used. This was followed by rapid improvement, and ultimately complete recovery. Bacteriological tests had shown that the first serum was weakly effective but that the second serum was many times more potent toward this particular organism.

In Table III are shown brief protocols of further illustrative cases in infants:

It is apparent that the recognition of meningococcus meningitis in infants is not easy, at least in the early stages. In New York City 28% of 627 cases were under 1 year of age[8], while in Massachusetts only 16% of the reported cases are in this age group[9]. One cannot escape the inference that not only are many of the cases of meningococcus meningitis not diagnosed until late in the disease, but that many escape detection entirely.

*Summary:* In a small series of hospital cases the fatality rate of infants with meningococcus meningitis ranged from 75 to 100% in those patients sick longer than one week before the disease was recognized and treated. Intensive treatment with injection of serum alternately

TABLE III

| Age | Symptoms and Duration before Treatment | Course |
|---|---|---|
| 7 wks. | Fever, convulsion—1 day. | Prompt response to serum. Sequel—iridocyclitis and loss of one eye. |
| 4 mos. | Fever, vomiting for 7 days, then stiff neck and convulsion. | Prompt response to serum. Complete recovery in 4 days. |
| 4 mos. | Irritability and vomiting, 3 days. | Complete recovery. Total duration 10 days. |
| 5 mos. | Fever, vomiting, drowsiness, irritability, stiffness of arms—3 days. | Prompt recovery in 5 days. |
| 11 mos. | Fever, anorexia, vomiting, loss of wt., irritability, drowsiness—3 wks. | Uneventful recovery after 4 days of treatment. |
| 2 mos. | Fever, failure to nurse, irritability, 4 days. Convulsion on 5th day. | Progressive process—death in 10 days, despite lumbar and ventricular treatment. |
| 6 mos. | Fever, irritability and vomiting — 11 days. Stupor on 12th day. | Death after 7 days of treatment. Serum into spine 4 times and into ventricle 4 times. |
| 4 mos. | Fever, diarrhoea, anorexia, vomiting, 2½ wks. drowsiness and irritability, .2 wks. Stiff neck, 5 days. | Intensive treatment produced little effect in cell count and organisms. Death in 5 days. |
| 5 mos. | Vomiting and irritability, 2 mos. Convulsions, 6 wks. Enlarging head, 5 wks. | Thick pus obtained. Death after 6 wks. treatment. Meningococcus abscess in rt. frontal lobe at necropsy. |
| 2 mos. | Vomiting, irritability, fever, failure to nurse—5 wks. | Persistent advancing infection. Death after 4 wks. of treatment. Serum into ventricles, spine, cistern, and basal operative opening. |
| 8 wks. | Fever, irritability, anorexia, 2½ wks. Convulsion 17th day. | Progressive basilar meningitis. Death after 5 wks. of treatment. Walled off pus found in left ventricle after 4 wks. |
| 11 mos. | Fever, vomiting, 19 days. Stiff neck, irregular respirations, 20th day. | Progressive chronic meningitis, operated at base of skull, no effect. Death in 3 wks. |

into the ventricles and lumbar subarachnoid space frequently succeeded in clearing up the infection, but did not alter the final outcome. Death in these latter cases was due to chronic hydrocephalus.

In contrast to this high mortality in late cases, the infants treated within the first week of the disease showed a percentage of deaths as low as that found for older children.

The necessity of wider attention to early diagnosis of the disease in infants or the development of more effective therapeutic procedures in late cases is obvious.

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# EXPERIMENTAL COMBINED SYSTEM DISEASE*

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THE appearance of certain abnormal neurological signs, characterized by a greater or less degree of spastic ataxia, is frequently encountered in Addisonian pernicious anemia, as well as in other conditions in which there is likewise a gastric anacidity. This clinical state appears to depend upon degenerative changes partly in the peripheral nerves but mainly in the spinal cord, consisting of a diffuse loss of myelin sometimes affecting chiefly the dorsal and lateral columns[1], and therefore known as combined system disease. It also has been observed in pellagra[2], certain cases of sprue[3] and even in the absence of manifest pathology, other than gastric anacidity[4,5]. On the contrary, this condition is only rarely encountered in cases showing a normal secretion of hydrochloric acid in the stomach[6].

The quantitative nature of the response of pernicious anemia to liver feeding, originally shown

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by Minot and Murphy[7], strongly suggests a deficiency disease[8,9]. The probability that the defective secretion of the stomach of these patients operates to produce a virtual deficiency in the presence of a diet adequate for the normal individual has recently been demonstrated by one of us (W. B. C.[10]). It was then shown that an interaction of normal human gastric juice with beef muscle under suitable conditions can produce a substance capable of effecting prompt improvement in patients with pernicious anemia in a manner entirely comparable to the action of moderate doses of kidney, liver or liver extracts[11]. Since the latter substances will promptly relieve not only the anemia of these patients, but, in a majority of cases, if regularly given in sufficient dosage for some time, will also greatly improve the neurological symptoms[12, 13], it appeared possible that some type of deficiency might also be the basis of the associated nerve changes. It could be supposed that the defective gastric secretion of the patients with pernicious anemia, which was apparently a factor in permitting the anemia to develop, might also be responsible in some way for a deficiency leading to the development of the combined system disease.

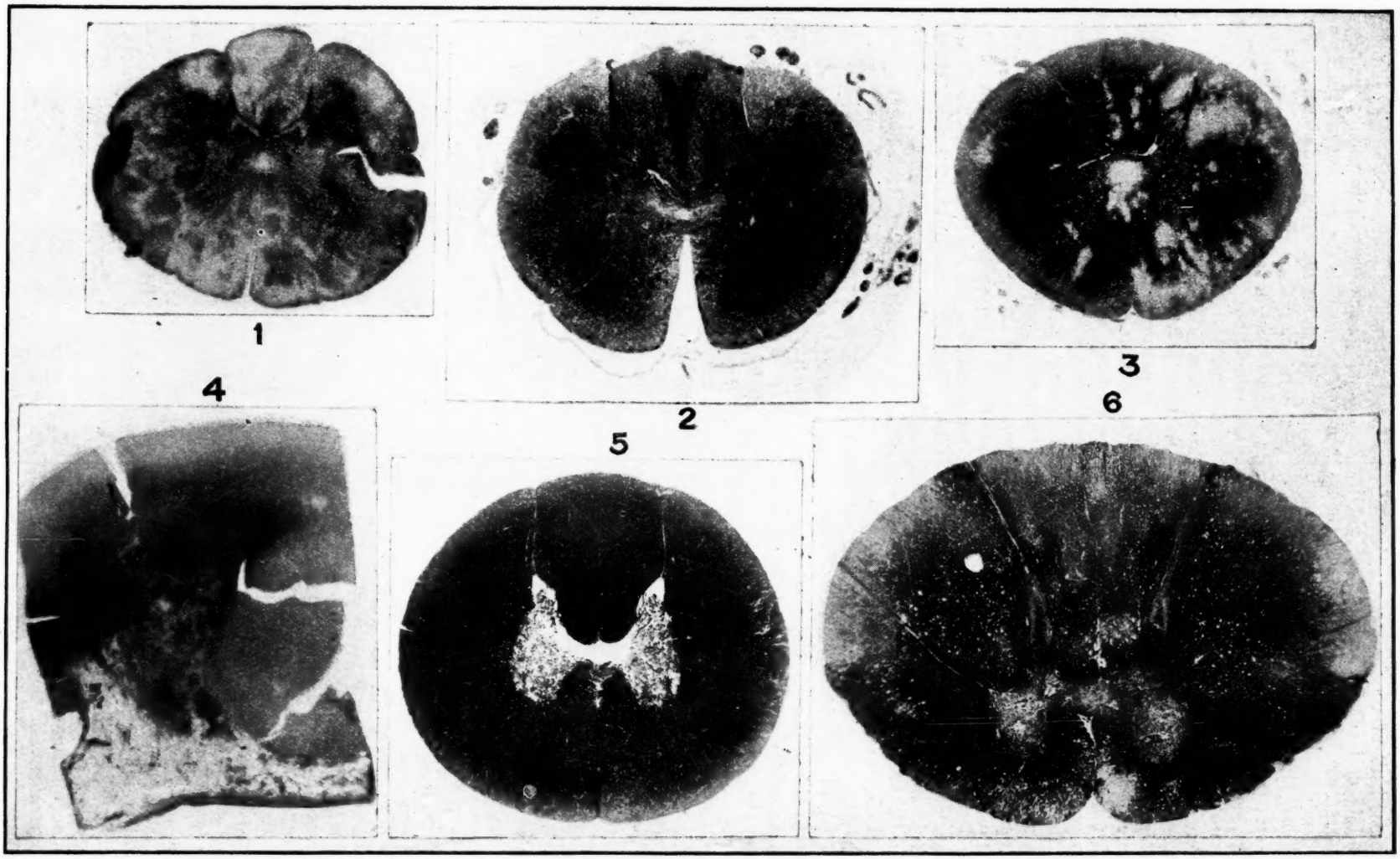
The effect of a lack of the antineuritic portion of vitamin B on the nervous system of animals has been clearly established. In beri-beri[14] and lathyrism[1], both of which are diseases probably due to a lack of this vitamin, cord lesions suggestive of combined system disease have been observed. Various investigators have reported changes in the gastro-intestinal tracts[15] and reduction of gastric secretions[16] in animals fed diets of varying kinds in which a deficiency of vitamin B was prominent. Boyenval[17] states that the daily injection of histamine, subcutaneously, apparently prevented the usual neurological symptoms, and the experiments of Dansye-Michel and Koskowski[18] correlated this effect of the histamine with the maintenance of normal gastric secretion in animals fed diets deficient in vitamin B. It seemed, therefore, worth while to attempt a critical study of the effect of a deficiency of vitamin B, in particular the antineuritic portion, upon potential changes in the gastric secretion and, perhaps, as a result, in the nervous system. Because Cowgill[19] had reported, in dogs fed diets deficient in vitamin B, the development of gastro-intestinal symptoms and spastic disturbances of locomotion, possibly resembling the symptoms of human combined system disease, it was decided to repeat, with particular attention to central nervous system changes, the experiments which he had described.

In March, 1928, the first group of eight dogs, series I, was placed on the diet used by Cowgill, which was probably deficient in both the antineuritic and pellagra preventing factors of vitamin B, and has been demonstrated by McCollum and others to be, in other respects, adequate. A second group of six dogs, series II, was given an identical diet except for the addition of autoclaved yeast to supply only the pellagra preventing factor of vitamin B. A third group, series III, composed of four dogs, was fed on a modification of a diet which Goldberger[20] has demonstrated to be deficient in the pellagra preventing factor. In view of the fact that Hofmeister[21] had shown that animals fed on diets completely deficient in vitamin B sicken and die rapidly without showing marked symptoms of paralysis, an effort was made to produce a chronic partial deficiency of the vitamin by feeding the animals small doses of vitamin B extract whenever they seemed to be on the verge of death. On this régime some animals lived from 100 to 200 days and showed repeatedly a prompt relief of symptoms on administration of vitamin B. Dogs in series I and II developed severe symptoms between the 60th and 80th days of this régime. These consisted, as described by Cowgill[19], of loss of appetite and weight, progressive weakness and stiffness of the legs, spastic paralysis, convulsions, coma and death. It was observed, however, that peripheral reflexes were ordinarily present even with marked paralysis, and the actions of the animals at times suggested a disturbance of position sense. Tenderness over the nerve trunks was never elicited. In a few animals rhythmic jerking movements of one or more extremities appeared. Until the last these symptoms could always be improved within a few hours by the administration of a few cubic centimeters of certain vitamin B extracts.* The dogs of series III lost weight and appetite, became very mangy, developed scaling skin lesions, and finally died in convulsions, without showing severe paralysis or spasticity.

The secretory activity of the stomachs of these animals was tested, at intervals of a few days, by determining the response to the subcutaneous injection of 0.5 milligram of histamine[22]. Free hydrochloric acid and pepsin were found in all satisfactory samples obtained half an hour after this procedure. No changes of a progressive sort or even significant alterations of the secretions of the paralyzed animals were observed, and no decrease of neurological symptoms once developed was obtained by repeatedly injecting histamine at intervals of a few hours. In some animals a slight degree of a secondary type of anemia developed.

Parts of brains and cords were fixed in formalin and in Müller's fluid, and stained with hematoxylin and eosin, cresyl violet, Scharlach R, and by Weigert's and Spielmeyer's techniques. The Marchi and Scharlach R stains were used on the peripheral nerves. In dogs of series I and II, the most marked lesions were found in the cord (Fig. 1) and next in the cerebral cortex. The peripheral nerves were least affected. Lesions

*Yeast vitamin B extract (Harris); and an alcoholic extract of wheat embryo, kindly supplied by Dr. G. H. A. Clowes of the Research Laboratories of Eli Lilly and Co.




Low power photomicrographs of degenerative changes in the central nervous system of dogs fed diets deficient in vitamin B. Weigert's[1] and Spielmeyer's stains.

Figs. 1, 2, and 3. Sections of spinal cords from three dogs of series I showing in all patchy loss of myelin in the white matter. In Fig. 2 the lesions are fairly symmetrical and most marked in the dorsal and lateral regions.

Fig. 4. Section from brain of a dog from series I showing an area of loss of myelin in the white matter below the cerebral cortex.

Fig. 5. Section of spinal cord of normal dog.

Fig. 6. Section of spinal cord of patient with pernicious anemia and symptoms of combined system disease showing diffuse loss of myelin in the white matter, mainly of dorsal and lateral regions, but with irregular patches elsewhere.

of varying degree in the central nervous system were present in all of the fourteen animals of series I and II and consisted of diffuse yet irregular loss of myelin, with patches of almost complete demyelinization. The edges of such patches shaded gradually into almost normal white matter. In these border areas most of the nerve fibers had irregular swellings that gave them a beaded appearance. Such changes were most obvious in the Spielmeyer stained sections. No evidence of an inflammatory process or of glial proliferation could be found in any of these dogs. Little or no fat was seen in the perivascular or meningeal spaces of the cord by Scharlach R stain, but a considerable amount was found in the perivascular spaces of the cerebral cortex and in some of its nerve cells. Cresyl violet stains brought out swelling and chromatolysis of the nerve cells of the cord and cortex. Scharlach R stains of peripheral nerves showed patchy degeneration of myelin in nine of the fourteen animals of series I and II much less severe than in the cord and cortex. A more detailed description of these changes will be published at a later date. Only minor degrees of the lesions found in animals of series I and II were found in the dogs of series III.

It may reasonably be concluded from these experiments that in dogs vitamin B deficiency, probably of the antineuritic portion, if sufficiently prolonged, produces a demyelinization chiefly of the white matter of the spinal cord and cerebral cortex and, to a less extent, of the peripheral nerves. The spastic paralysis of these animals was consistent with the presence of predominating lesions of the central nervous system. Previous workers seem to have stressed the importance of lesions of peripheral nerves and have not observed or sufficiently emphasized the cord pathology[23, 24]. This may be due to the fact that in our animals the deficiency was made as chronic as possible. In human beri-beri, similarly, the emphasis seems to have been placed upon the peripheral neuritis, although cord lesions have been observed[14, 25]. It is interesting that the pathological lesions in the cords of these animals closely resemble those found in the so-called combined system disease, which, in man, has such a constant association with achlorhydria and is so frequently encountered in cases of Addisonian pernicious anemia.

Since no significant changes in the gastric secretions of these animals were observed, no dependence upon achlorhydria for the production of these lesions was demonstrated. It is possible, however, that, as is probable in pernicious anemia[11], the absence of some factor, other than the acid of the normal gastric secretion, is responsible for the changes seen. Nevertheless, the experiments of Boyenval[17] and others[18] with histamine were not confirmed by these observations nor by other attempts made by us to repeat their experiments. No support therefore, is lent to the attractive hypothesis that histamine or vitamin B are essential for maintaining a normal gastric secretion which, in turn, in some way preserves the nervous system from the injury which results from a deficiency of vitamin B in the food.

The production by a deficiency of vitamin B, particularly the antineuritic portion, of lesions in the spinal cord of these dogs, closely resembling those of human combined system disease, suggests, however, that the basis of this condition in man may be a deficiency of the antineuritic portion of vitamin B. It is possible that the achlorhydria is the factor which in some way conditions a virtual deficiency of this portion of vitamin B for such patients, just as it may apparently produce a deficiency of iron for certain cases of anemia[26,27]. Consequently, this possibility is at present under investigation and some evidence has been obtained in this clinic that, by giving large quantities of vitamin B to patients suffering from combined system disease, improvement in their symptoms may be observed.

## SUMMARY

1) Eight dogs fed a diet deficient in both the antineuritic and pellagra preventing portions of vitamin B, and six dogs fed a diet deficient only in the antineuritic portion developed loss of appetite, vomiting, a spastic type of paralysis, beginning usually in the posterior extremities and progressing rapidly to convulsions, coma and death, unless treated with vitamin B extracts. Four dogs fed a diet lacking mainly the pellagra preventing portion of vitamin B developed no significant neurological symptoms, but died eventually in convulsions.

2) Post mortem examination showed that all fourteen of the animals chronically deprived of the antineuritic portion suffered from a diffuse, irregular loss of myelin chiefly in the white matter of the spinal cord, less marked in the cerebral cortex. In nine of these animals slight lesions of the peripheral nerves were found. Early changes were observed in the nervous systems of the four dogs placed on the diet deficient in the pellagra preventing portion of vitamin B.

3) The lesions in the spinal cords of the animals of the first two groups were considered to bear a close resemblance to those encountered in human combined system disease.

4) Gastric analyses showed no significant changes in the course of these observations and thus no evidence for a causal relationship between combined system disease and achlorhydria was established.

5) Since the chronic deficiency, particularly of the antineuritic portion of vitamin B, appeared to produce in the dogs lesions closely resembling those occurring in human cases of combined system disease in which achlorhydria is almost invariably encountered, it is suggested

that the basis of the development of that disease may be a virtual deficiency of the antineuritic portion of vitamin B brought about in some way by the achlorhydria.

We are greatly indebted to Dr. Stanley Cobb for suggestions and opinions referable to the pathological histology, and to Dr. George R. Minot for information concerning unpublished observations of the effect of vitamin B on the cord lesions of certain cases of human combined system disease.

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# THE GOVERNOR'S WELFARE PROGRAM AND CERTAIN ASPECTS OF CHRONIC DISEASE*

BY ROBERT B. OSGOOD, M.D.†

HIS Excellency Governor Allen has recommended to the two branches of the Legislature of Massachusetts that an intensive study of the problem of aged sufferers from chronic disease be made "with the particular purpose of determining how far the insecurity of old age may be lessened by early diagnosis and treatment of cases of chronic disease."

I welcome the opportunity of speaking before this committee of citizens to strongly urge favorable action upon these recommendations. My own professional work happens to be largely concerned with the chronic sick. The appeal they make is an intense one. Early causes of their chronicity are being glimpsed. Early diagnosis is increasingly possible. Early treatment is becoming more efficient. Much may be done to lessen the handicap of even the advanced cases. But facilities are necessary for this possible betterment. No harder lot exists than that of the aged chronic sick when these facilities are lacking.

Probably the first object of an investigation such as His Excellency has recommended would be to ascertain what is being done at present for the cases of chronic illness in Massachusetts. The second object would be to ascertain whether adequate facilities already exist for early diagnosis and treatment. If adequate facilities do not exist, the third object would be to determine how best to provide such facilities.

The report of the combined Social and Medical Committees of their Survey of Chronic Disease in the City of Boston made in 1927 is grist to our mill when we attempt to answer these questions.

This Survey revealed the fact that though chronic diseases are common attendants of old age, youth and middle age also suffer seriously from chronic disease: 58 per cent. of the 4,316 cases this Survey reviewed affected people under 60 years and 42 per cent. people over 60 years. In October 1927, there were found in all Boston hospitals whose work was not solely confined to acute cases, over 700 chronic or incurable cases occupying their beds. Of the 4300-odd cases of chronic disease, 85 per cent. had been ill over three months and 50 per cent. had been ill over six months. The Survey suggests that many chronic patients can be adequately cared for in their own homes and that they strongly prefer to be so cared for, if varying amounts of financial help can be provided by some private or public agency. Such aid would

*Read at the meeting on Governor Allen's Welfare Program, February 3, 1930.

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solve a part of the problem for the incurable and for the partially disabled after they had been studied and rehabilitated to their fullest extent.

It is significant, however, that on October 19, 1927, which was taken as a Hospital Census Day, there were among some 3,000 patients in the Boston Hospitals for acute and general medical and surgical illness, over 500 cases who still required treatment for over three months. It appears, therefore, that in our general hospitals for acute sickness, 1/6 of their population consists of cases which may be considered as chronic. The vast majority of these cases were probably Boston cases and in this city there are less than 1,000 beds available for chronic cases. On October 19, 1927, there were more than half this number of cases occupying beds in Acute Hospitals who needed Chronic Hospital treatment. Exclusive of the 450 beds at Long Island, facilities are seriously lacking for the adequate care of chronic cases in the City of Boston. This lack is actual and even exaggerated because self-respecting chronic patients quite naturally hesitate to accept hospitalization at Long Island. They wrongly but persistently think of this excellent institution as an almshouse for incurables tenanted solely by the pauper class. There are probably only half the number of beds available in Boston for patients who are able to contribute something toward their hospital support. The case work agencies reporting to the Survey concluded that the greatest and most constant need of the aged chronic sick is for hospitalization at a rate within the means of the patient or his friends. Nearly 500 cases needing nursing and medical care away from their homes were discovered by the Social Agencies in Boston and this number would undoubtedly be greatly increased if information had been obtainable as to cases under the private care of physicians. Conditions throughout the state are probably no better than those in Boston. If hospitalization cannot be provided, many of these patients must seek for care in the so-called nursing homes which take what they can get and give only in proportion. They are a poor makeshift for kindly and purposeful hospitalization and their conduct is hard to control.

A specific instance of a specific need may not be amiss.

To refer again to the Boston Survey, the four most prevalent chronic disabilities were found to be heart disease with 845 cases, organic mental and nervous diseases with 553 cases, cancer with 534 cases and chronic arthritis with 467 cases. If one analyzes the duration of these various disabilities, it is found that about 21 per cent. of the heart cases had existed for over 5 years and only 8 per cent. for over 10 years; 31 per cent. of the mental cases had existed over 5 years and 14 per cent. over 10 years; 8 per cent. of the cancer cases had existed over 5 years and only 3 per cent over 10 years. But 44 per cent. of the arthritic cases had existed for over 5 years and 25 per cent. for over 10. These long disabilities from arthritis, therefore, total more than ½ of all the other three prevailing chronic diseases combined for the 5 year period, and nearly ¾ of all the others combined for the 10 year period. The arthritic cases largely outnumber the long period disabilities of any of the more prevalent chronic diseases and when one considers their helplessness and their need of attendance, they perhaps become more important economically than any or all of the other common chronic disabilities. If one considers the whole rheumatic group and includes the chronic heart lesions caused by rheumatic fever, the problem of non-tuberculous joint disease would seem to bulk larger than any of the other causes of ill health of the aged or of those in middle life.

The solution of the problem evidently and hopefully lies along the lines of early diagnosis and adequate treatment as the Governor suggests. Both early diagnosis and adequate treatment in a very large percentage of cases will be possible and efficient if the medical conscience can be awakened, the lay public informed and more adequate hospitalization made available.

England and Sweden are awake to the waste of chronic rheumatism, Germany and France are waking, and America slumbers in spite of voices crying in the night, but alarm clocks are set and ticking and the American Committee for the Control of Rheumatism, a branch of the International Committee, is hard at work. They are to a man optimistic as to the possibility of recognizing early signs and as to the possibility of controlling the disease when early diagnosis is made and adequate treatment can be provided. Taking out tonsils and teeth does not cure the chronic sufferer. There is no rapid transit for the arthritic to the goal of health, but the path is becoming clearer and more and more cases reach the goal. They must have rest and exercise, sunshine and appropriate food. They must have skilled diagnosis checked up by laboratory findings. They must have local treatment of their joints by physiotherapy and apparatus, and when there are fixed deformities, by surgery. By combinations of simple and comparatively inexpensive methods, very worth while amounts of rehabilitation and the arrest of the disease are possible. The records of the Robert B. Brigham Hospital prove this statement.

I plead for the sufferers from chronic rheumatism and for the prevention of crippling arthritis. Others have pleaded for other important chronic diseases.

His Excellency the Governor is right,—an intensive study should be made of problems of continuing disability. Adequate facilities for their care and betterment appear to be lacking in Massachusetts. We believe the insecurity of old age may be lessened by the early diagnosis and treatment of chronic illness.

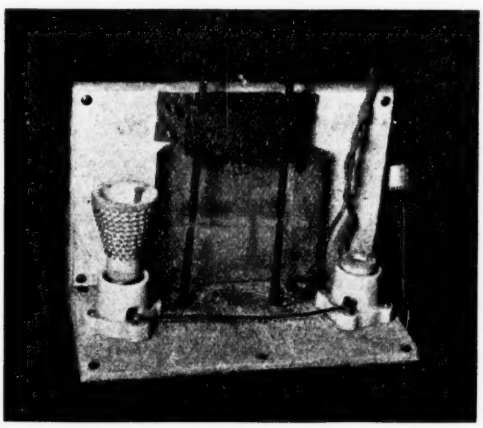
# NEW ENGLAND PEDIATRIC SOCIETY

## A SIMPLE THERAPEUTIC ARC

BY EUGENE C. PECK, M.D.*

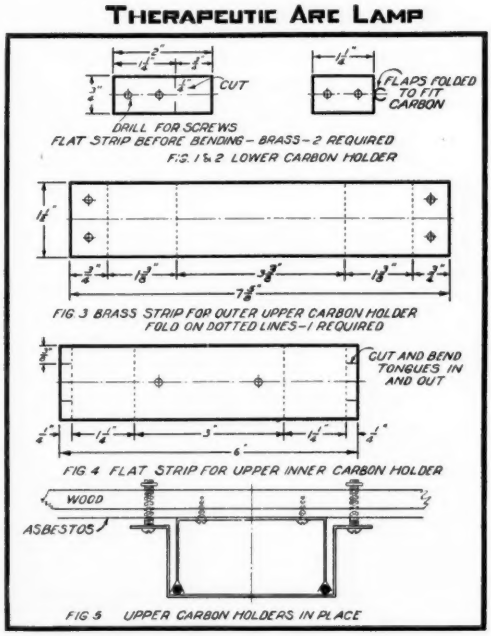
THIS arc lamp is worthy of description because it has been so simplified that any amateur mechanic can make one in a few hours at a slight cost. It is electrically and mechanically effective in producing a pair of arc flames between two pairs of therapeutic carbons using 660 watts of power. From this model others are easily designed utilizing any amount of power which it is safe to draw from household wiring.

In designing an arc lamp the fundamental nature of it must be kept in mind. It is an apparatus holding carbon pencils in such a position that an arc may be easily formed between their points and the current will continue to pass at the proper amperage. The electrical formula used is I = E/R or the size of the current (Amperes) equals the tension (Volts) divided by the resistance (Ohms). Each arc flame has a drop of voltage of 30 to 40 volts across it. In this type of lamp two arcs and the resistance are in series and the voltage drop in each of the three parts may be assumed to be 36 volts, total 108 V. Thus the amperage passing through the lamp will be 36 divided by the ohms in the resistance unit. Different resistances could be used with corresponding change in effect. The rule is that under the same conditions the amount of light varies as the square of the amperage. Many types of carbons are for sale, the nature of the light depending upon the elements contained in the core of the carbon pencil and so volatilized in its flame.



The small lamp which I have used for nearly two years has a 6 ohm resistance and so the amperage is also six. The resistance unit used in this home made lamp is made from an ordinary 660 watt radiator cone by removing 2/3 of the wire and stretching the remainder to fill the grooves in the porcelain. The circuit is from the 110 volt line through a socket F containing a 10 ampere fuse, through a socket R containing the resistance element, to the lower carbon C1 on one side, up across the first arc to the upper carbon C2, across the brass holder B of the upper two carbons to the second upper carbon C3, down across the second arc to the lower carbon C4 and to the power line. The whole is mounted on a base of wood 7″ x 7/8″ x 12″ with a back 7″ x 7/8″ x 10″. The wood is well protected by asbestos board.

The carbon holders are made from soft brass 1 and 1/2″ wide and 1/32″ thick. The diagram shows the dimensions and bending of the


THERAPEUTIC ARC LAMP
CUT
FLAPS FOLDED TO FIT CARBON
DRILL FOR SCREWS
FLAT STRIP BEFORE BENDING – BRASS – 2 REQUIRED
FIG. 1 & 2 LOWER CARBON HOLDER
FIG 3 BRASS STRIP FOR OUTER UPPER CARBON HOLDER
FOLD ON DOTTED LINES – 1 REQUIRED
CUT AND BEND TONGUES IN AND OUT
FIG 4 FLAT STRIP FOR UPPER INNER CARBON HOLDER
WOOD
ASBESTOS
FIG 5 UPPER CARBON HOLDERS IN PLACE

parts. The lower holders are bent up and shaped around a carbon, then the carbon removed and the brass socket compressed slightly so that the carbon will fit tightly into it. The holder of the upper carbons is two pieces. The inner piece has three cuts 1/4″ deep from each end making four small tongues which are bent alternately right and left to allow the carbons to rest in the Ys thus formed. This inner piece is fastened with wood screws to the back near the top so that its lower edge is 5″ from base. The outer piece is fastened loosely around it by 2″ machine screws which go through the wooden back. The machine screws are tightened carefully so that the outer piece presses gently on the carbons, and they can be easily pushed

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up or down but will not slip. This piece does not touch the asbestos of the back. It is the correct adjustment of the pressure of this outer piece on the carbons which enables them to be controlled easily by hand instead of by the expensive mechanisms found in the commercial lamps. Below this upper fixture is a reflector made of a piece of polished aluminum 4 and 1/2″ x 6″. The lower carbon holders are placed directly under the places of the upper carbons. The connections between parts are made with bare wire or bus bar.

An electrician or mechanic can make improvements in it but I strongly advise using this design unless the worker is very expert. I have tried many improvements which have been suggested with little success. Recently, however, I have made some lamps with two resistance sockets arranged in parallel, so that I can put two units in the circuit. If then each of these has 7.2 ohms resistance the effect is like 3.6 ohms and a 10 ampere arc results. I have not used these long enough to know how well they will stand up but I think they will be very useful for larger children. The same time exposures scheduled for 15″ with the 6 ampere lamp can be given at 25″ with the 10 ampere lamp.

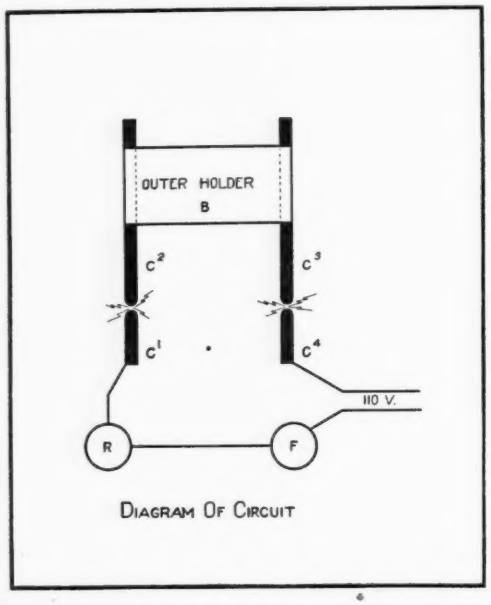

Diagram Of Circuit

The carbons for the best ultra-violet effect are 6 mm. 6″ therapeutic "C" carbons. A short piece is placed in each of the lower holders and whole carbons in the upper. When the two upper carbons are pushed down to touch the lowers a glow appears at the contact point. They are then pulled apart a very little and the arc established. While the arc is burning the carbons shorten so that the arc goes out in about one and a half minutes if untended. By gently pushing or tapping the upper carbons are fed downward to maintain the arc during the treatment. After the lower pieces are consumed the upper carbons are transferred to the lower holders. Always keep a small pair of pliers by the lamp for handling the carbons and adjusting the holders. Of course the current should be turned off while changing carbons or making adjustments but you will be surprised to find the upper carbons can be handled with impunity while the current is on.

The usual orders for the use of the lamp are:

1. C carbon arc 6 amp. 15″ from light to skin.
2. Always protect the eyes. Beware of hot or electrically charged parts.
3. Five minutes the first day to each surface exposed. Treat daily, increasing one minute each day until mild sunburn appears. Then increase one minute every other day until ten minutes is reached.

The usual tonic treatment of a baby is ten minutes to the front and ten minutes to the back three times a week. This keeps a mild tan on the trunk and effectively prevents rickets. I have also used the lamp for its combined infra-red and ultra-violet effect on presistent discharging ears, cervical adenitis, persistent septic discharging surfaces, and furuncles, with results which the parents and I believe beneficial. But it is only an adjuvant. No useful therapeutic procedure should be neglected.

The principal object in presenting this apparatus to you is to show how very simple and inexpensive an effective lamp can be. If such models are common they will tend to counteract the excessive commercialization of the benefits of ultra-violet light or artificial sunlight which is so obnoxious to most of us. This lamp is no better than the commercial lamps but it is so easy to make that any intelligent father can make one for about the same cost as one ultra-violet treatment at the office.

List of parts used in building a 6 ampere lamp.

1 board 7″ x 7/8″ x 22″.
1 piece asbestos board 10″ x 12″.
2 porcelain base sockets.
1 Radiator element 660 watts with screw base.
1 piece aluminum 4 1/2″ x 6″ (Cheap cake pan can be cut).
1 piece soft brass 1/32″ x 1 1/2″ x 16″.
4 brass machine screws 2″ long with nuts to fit.
Screws, wire, and electric connecting plug.
"C Therapeutic" carbons 6 mm. x 6″. Can be purchased only on a doctor's prescription.

# CHART FOR THE COMPUTATION OF HARRIS-BENEDICT STANDARDS OF BASAL METABOLISM

BY CURTIS BRUEN, M.D.*

THE normal basal metabolism per day is predicted by the Harris-Benedict method from the regression equations

For Men

$$h = 66.4730 + 13.7516\ w + 5.0033\ s - 6.7550\ a$$

For Women

$$h = 655.0955 + 9.5634\ w + 1.8496\ s - 4.6756\ a$$

in which $h$ represents heat-production in calories, $w$ weight in kilograms, $s$ stature in centimeters, and $a$ age in years[1].

section, and reading off its value. Separate, generally symmetrical, contiguous charts are constructed for men and women. The limits of age, height, and weight are adapted from the scatter diagrams in which the series of these data from which the equations are derived are plotted against daily heat-production[1]. In each chart the $x$ and $y$ axes are divided in caloric units to such a scale and over such a segment as is required by the scale equations for the ranges assigned the three variables. The corrective co-efficients for stature and weight are

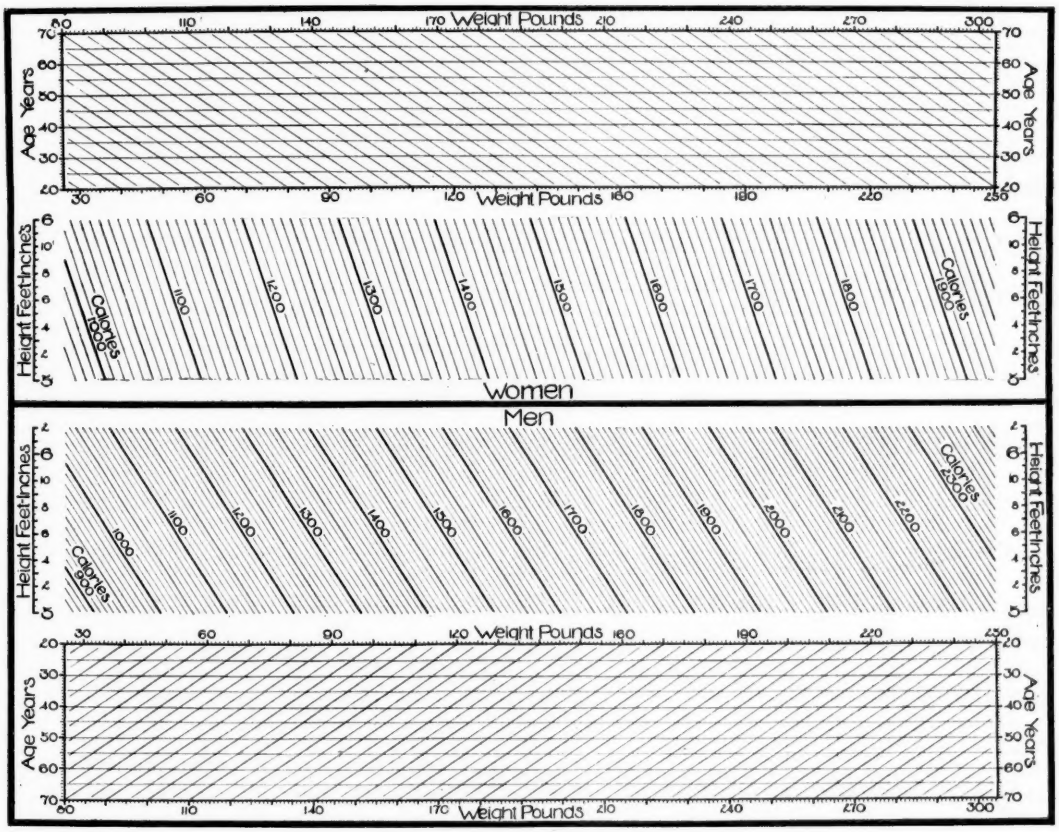


CHART I. Basal metabolism per day (Harris-Benedict).

The resultant standards of basal metabolism are computed in multiple prediction tables[1]. Each equation requires two complementary tables. The first gives its constant term plus the corrective term for weight. The second gives the algebraic sum of the corrective terms for stature and age. The algebraic sum of these two factors gives the basal metabolism.

This result can be obtained graphically by locating two coördinates, sighting their intermultiplied by 2.5400 and 0.4536 respectively. The scales of height in inches are marked off on the $y$ axes by plotting the corrective terms for height computated from the scale equations

For Men

$$y = 12.7084\ s_{in.}$$

For Women

$$y = 4.6980\ s_{in.}$$

The combined scales of age in years and weight

*Bruen—See "This Week's Issue," page 548.

in pounds plotted on the $x$ axes from the equations

For Men

$$x = 66.4730 - 6.7550\ a + 6.2377\ w_{lb.}$$

For Women

$$x = 655.0955 - 4.6756\ a + 4.3380\ w_{lb.}$$

form a network. Separate weight scales a suitable distance apart are plotted for the limiting ages. The constant term of each equation minus the corrective term for the minimum age is laid off on one, the constant term minus the corrective term for the maximum age on the other. These scales are marked off by laying the corrective terms for weight off from these points. Lines connecting the termini of each pair are divided in equal parts to form age scales. At intervals points of equality on the opposing scales of age and weight are connected by straight lines. The joint coördinate for age and weight is thus the intersection of projections from their separate scales into the network. Graphs are drawn for series of equations

$$h = x + y$$

Chart I computes the daily basal metabolism for each sex from age, height, and weight. Go to the upper section for women, to the lower for men, spot the age in years, carry an imaginary line horizontally into the network, spot the weight in pounds, carry an imaginary line in the direction of the inclined guide lines to its intersection with the horizontal line previously traced, from this point carry an imaginary line vertically into the field of the chart, spot the height in feet and inches on the nearest scale, carry an imaginary line horizontally to its intersection with the vertical line previously traced, estimate the heat production in calories by interpolating between the adjacent graphs.

REFERENCE

1 Harris, J. A., and Benedict, F. G.: A Biometric Study of Basal Metabolism in Man. Carnegie Institution of Washington, Publication No. 279, 1919.

## CO-OPERATIVE RURAL HEALTH WORK OF THE PUBLIC HEALTH SERVICE

A report of the co-öperative rural health work of the Public Health Service for the past fiscal year has recently been issued. This report is of especial interest. This work is conducted in co-öperation with State and local health authorities and is made a part of a well-rounded comprehensive program of local (county or district) health service.

At the beginning of 1929 there were 467 whole-time county or district health officers in the United States. The principle of co-öperative rural health work appears sound in theory and successful in practice. State health departments, in increasing number from year to year, are obtaining authorization and appropriations to enable them more nearly to do their proportionate parts in the development and maintenance of full-time county health service.

Under this co-öperative arrangement the rural sanitation work of the Public Health Service is carried out in each project by a local health force intended to be permanent and is made a part of a general program of rural health work deemed suitable to the locality. Thus, it is accomplished more economically and with more lasting effects from a demonstration standpoint than it could be if undertaken by a specialized force working a comparatively short time in the locality.

The unit for the work, as a rule, is the county, but it may be a group of townships in the same vicinity or a district comprising two or three adjacent counties. In some of the units incorporated villages, towns, and cities (with populations of approximately 50,000) are included. Under the co-öperative arrangements a good program of health work can be carried out in practically any rural county or district in the United States at a cost to the county or district easily within its means. The average co-öperative demonstration project is conducted on a cost basis of less than fifty cents per capita of population served and furnishes a striking example of efficiency with economy in public service. In many counties efficient whole-time county health service can be provided at an annual cost of less than two dollars to the local taxpayer with real property assessed at $5,000 to $6,000.

An annual budget of $10,000 will provide in most sections of this country the services of a county health department force consisting of one whole-time health officer, one whole-time sanitary inspector, one whole-time health nurse, and one office clerk. Such a force can render highly effective health service in any county with a population under 25,000 and an area under 500 square miles. For larger units of population, larger forces are needed and should be provided, especially after the first year or two of operation.

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## CANCER INCREASE CONTINUES

The January, 1930, *Statistical Bulletin of the Metropolitan Life Insurance Company* states that the cancer mortality rate among its policyholders has now reached a new high point—its maximum rate since 1911. The 1929 rate was 77.3 deaths per 100,000 policyholders, a figure 13.7 per cent. in excess of the 1911 rate. The most discouraging fact in their investigation was that there was no evidence of a significant decline in the deathrate for cancer of any organ or part of the body.—*Bulletin American Society for the Control of Cancer.*

# MEDICAL PROGRESS

## PROGRESS IN PEDIATRICS

BY JOHN LOVETT MORSE, M.D.*

INSTEAD of attempting to go over all the literature of Pediatrics during the last year, which, by the way, is abstracted most completely in the *American Journal of the Diseases of Children*, I have selected three subjects, which seem to me the most interesting at the present time, for discussion. They are "Nephrosis", "Acute Pyelitis" in Infancy, and Hypoglycemia, Convulsions and Recurrent Vomiting.

### NEPHROSIS

It requires considerable temerity to approach this subject, about which my ideas are so hazy and about which others talk so glibly and seem to know so much. Christian gives a summary of the literature of the subject in an admirable article in the *Journal of the American Medical Association*, 1929, *93*, 23. Another very good article is that by Buck in the *New England Journal of Medicine*, 1929, *201*, 973. Altogether the best presentation of the pathology of this symptom-complex in children is that of Wolbach and Blackfan in the *Transactions of the Association of American Physicians*, 1929, *44*, 77.

It is extremely difficult to discuss the diseases of the kidney, or those involving the kidney, because neither physicians nor pathologists agree among themselves or with each other in their terminology, the great difficulty in determining from the symptoms and urine during life the pathological conditions which will be found after death, and the fact that many pathological conditions show the same urinary changes. In a very general way, as I see it, the pathologic changes in the kidneys in early life may be divided into inflammatory, affecting primarily the glomeruli, and degenerative, involving the tubules. The degenerative changes in the tubules may occur without any changes in the glomeruli, but very often are secondary to the circulatory changes in the glomeruli, both glomeruli and tubules being involved as the result of inflammatory lesions in the glomeruli. The degenerative lesions in the tubules may be the direct result of the action of known toxins and mineral poisons. The interstitial tissues of the kidneys in childhood are not involved primarily, except in acute suppurative nephritis—"pyelitis". Cicatricial changes in the tissues are, as a rule, the result of this disease or of reparative changes initiated by lesions of the tubules or glomeruli.

It is interesting to try to find out how the term "nephrosis" came to be used. About twenty-five years ago Muller suggested it for the degenerative changes in the tubules and is thus responsible for much of the confusion and discussion among the pathologists since that time. Epstein added to the confusion by his papers on Parenchymatous Nephritis and Edema in 1917. Munk, in 1913, described his lipoid nephrosis or "the kidney of syphilis". Others recognized that the same pathologic changes were caused by mineral poisons. Others apply the term to a rather characteristic symptom-complex, in which the changes found in the kidneys are chiefly degenerative. Still others say that this complex is simply our old friend, parenchymatous nephritis, in an edematous stage, and that all that is new is the name. There is certainly nothing new about the pathologic changes. The only ground for argument about them is, therefore, as to their etiology and significance. Still others wish to limit the use of the term "nephrosis" to those cases in which double refractive lipoids are present in the urinary sediment. This does not seem quite reasonable, in view of the statements of others that these are often present in the urine and not in the kidneys at autopsy and that they may be present in the kidneys and not in the urine. Furthermore, these lipoids are merely one of the stages in the degenerative changes found in the kidneys.

In spite of these differences of opinion, there does seem to be a fairly definite symptom-complex in children, at any rate, to which the term "nephrosis" may be applied, if one desires to use it. Whether this symptom-complex deserves a special name, whether nephrosis is the best one, if it does, or whether it should be considered now, as in the past, as simply one of the various manifestations of subacute or chronic nephritis, meaning by that term, disease of the kidneys, is a matter upon which opinions may, with our present knowledge, reasonably differ. The characteristics of this symptom-complex are: an insidious onset, marked edema, oliguria, and marked albuminuria with many cells and casts, but no red corpuscles. The presence of doubly refractile lipoid droplets is considered essential by some, but not by others, according to their conception and limitation of the pathologic changes. The basal metabolism may be diminished or normal, the blood pressure is not increased, the secretion of phenolsulphonephthalein is good, and there is no increase in the non-protein nitrogen of the blood. The blood proteins are diminished with a relative increase in the globulin, reversing the usual albumin-globulin ratio. There is also hypercholesteremia. Another characteristic is the tendency to remissions and exacerbations in the symptoms. Death very often occurs as the result of an infection

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with pneumococci or streptococci, which is usually localized in the peritoneum.

Wolbach and Blackfan report in detail the clinical histories and pathologic findings in eight cases showing this syndrome in childhood. Degenerative changes, varying from cloudy swelling to necrosis of the cells of the tubules, were found. In four of the cases small cicatrices were found, which were undoubtedly the end result of previous necrosis of the tubules. They state that in every instance the tubular lesions found represent the effects of an attack or exacerbation lasting from a few weeks to several months. In no instance was there cellular infiltration of the interstitial tissue of the kidneys, except immediately adjacent to necrotic tubular epithelium. They conclude that whatever degree of injury may have existed in the kidney at any one period, the effect is not cumulative.

Degenerative changes occur in the tubules in every acute febrile disease and are associated with more or less albuminuria. In some cases of diphtheria, for example, changes as marked as the most severe in these cases can develop in three or four days. These same changes may develop as the result of acidosis or of mineral poisoning. There is evidently nothing characteristic about the lesions found in the kidneys in these cases. When they are found without this symptom-complex they are considered to be secondary to disease elsewhere and due to something in the circulating blood which injures the cells. Recovery from such lesions is, at any rate, possible, and undoubtedly occurs except when death ensues as the result of the causative disease.

It does not seem unreasonable to consider, then, as do Wolbach and Blackfan, that this syndrome is not of renal origin, but a separate disease entity. While such a supposition is not unreasonable, it is a long way from being proved. It has been suggested by several that a disturbance of the function of the thyroid is the primary cause, because lesions have been found in it. Wolbach and Blackfan conclude, however, that the lesions found in the thyroid are simply manifestations of functional exhaustion and neither the cause nor the direct consequence of the kidney change. The same conclusion can be drawn as to the changes in the liver. Treatment with thyroid extracts by various men has, moreover, not proved satisfactory. Strange to say, no one has yet suggested, or, if they have, I have not seen it, that this symptom-complex may be due to allergy or an avitaminosis! Perhaps it is. Who knows?

Reasoning from analogy, it would seem that the most natural cause for such degenerative changes in the kidney would be a focus of infection somewhere in the body. Everyone has, of course, sought for them. Clausen (*Amer. Jour. Dis. of Children*, 1925, *29*, pp. 581 and 587) believes that the staphylococcus is the cause and that the focus of infection is most often in the nasal sinuses. He and others believe that they have cured some patients by treatment of the sinuses. Owing to the tendency to remissions in this condition, a longer time should elapse than in these cases before a cure is claimed. Incidentally, in two of the three cases reported by Wolbach and Blackfan, in which the mastoids and sinuses were examined, they were normal. The experience of most observers has been, I think, that it has not been possible to find a focus of infection. Nevertheless, it seems reasonable to believe that in a certain proportion of the cases presenting this syndrome, the cause may be a focal infection. If it is, why give it a special name, instead of looking on it simply as one of the manifestations or results of focal infection? The apparent cure of certain cases presenting this symptom-complex does seem, however, to strengthen the position of those who believe that the cause is not a primary disease of the kidney, but something outside of it.

If the degeneration of the renal tubules is not due to a bacterial toxemia or some mineral poison, it seems necessary to fall back on some disturbance of the metabolism or autointoxication as the cause. This may sound well, but means nothing definite. These terms simply serve at present as a cloak for ignorance. Nevertheless, an attempt ought to be made, I suppose, to determine what the changes in the blood suggest in this connection. It is safe to say that no one knows exactly what the function of cholesterol is. It is known to be increased in hydremia and when the blood protein is low. Both these conditions are present in "nephrosis". It is fair to presume, therefore, that the increase in the cholesterol in the blood is secondary to one of these conditions or due to the same cause. It is found in severe metabolic disturbances, notably diabetes mellitus. Its significance in this disease is not understood. It is evident, therefore, that the increase in cholesterol shows nothing definite as to the etiology of this symptom-complex. The presence or absence of lipoid bodies in the urine apparently depends on what stage the degenerative process in the kidneys happens to be. The blood proteins are low and the albumin-globulin ratio reversed, because of the loss of albumin in the urine. There is no evidence to show, however, whether trouble in the kidney is the cause of the loss, whether there is something in the blood which injures the kidney, making it more permeable, or whether there is something in the blood which makes the albumin pass more easily. Far be it from me to attempt to discuss the chemistry of the colloids and surface tension. Christian surmises that there is more albumin in the urine than can be accounted for by the lesions in the tubules and, therefore, that there must be a functional change in the walls of the capillaries of the glomeruli which allows the passage of albumin. The lack

of increase in the non-protein nitrogen and the approximately normal excretion of phenolsulphonephthalein simply indicate that the glomeruli are not involved. They show nothing as to any metabolic changes. Ruling out thyroid deficiency as the cause of the lowered basal metabolism, if it is lowered, there is no obvious cause for it, except the general nutritive disturbance. Certainly there is nothing to suggest that it is the basis of the trouble. It seems evident, therefore, that the changes in the blood show nothing as to the etiology of this syndrome. There being no evidence to show that there is any peculiar metabolic change to cause this symptom-complex and no focus of infection constantly found, the conception that it is not of renal origin can not be considered as proven.

Christian advances some of the arguments in favor of this symptom-complex, known as "nephrosis", being simply a form of chronic nephritis (Bright's disease). He summarizes and discusses the results of pathologic studies in adults, but says that the reports seem to indicate that the pathologic picture seen in those dying with an acute infection after a relatively short duration of life is due to the bacterial infection and is not what the kidneys would have shown if examined years after, as is the case in chronic nephritis. He suggests that perhaps many of the cases which are reported as cured are really not cured and that, if they were followed for many years, it might be found that the cures were only apparent. He calls attention to reported cases in which the clinical picture shifted from that of nephrosis to that of subacute or chronic glomerular nephritis and at death showed granular atrophied kidneys. He also quotes cases in which definite glomerular lesions, as well as degenerative changes in the tubules, were found in patients dying relatively early. He also calls attention to the cases which both clinically and pathologically present evidences of both nephrosis and nephritis and which gradually lose the clinical symptoms of nephrosis and take on those of glomerular nephritis. He refers to the work of Barker and Kirk, who, by keeping the blood protein of dogs low for a long time, produced in them edema, low basal metabolism, reversal of the albumin-globulin ratio and increased blood lipoids. He mentions a case of his own in which the autopsy showed degenerative changes in the tubules without any glomerular changes, but who had not shown the clinical picture of nephrosis.

Some of his conclusions are—"1. Given a considerable and prolonged protein deficit in the blood, edema, lowered blood protein, relative increase in blood globulin at the expense of albumin, hypercholesteremia and lowered basal metabolism will appear, not always in the same order and possibly in varying degree.

"2. The most common cause for such blood protein deficit is marked and prolonged albuminuria.

"3. The cause of such albuminuria is a renal lesion.

"4. Often this renal lesion is obviously in the glomeruli, but in many reported cases, mainly those patients dying of infection relatively early in the course of the disease, the glomeruli show very slight or no demonstrable lesions, while there are extensive degenerative changes in the tubules. For the latter, knowing what we do of renal physiology, and believing that the actual daily amount of albumin in the urine in many of these patients is in excess of what could possibly come from degenerating renal epithelium, I am of the opinion that the source of the albuminuria is in the passage of albumin through the walls of blood vessels, most probably, glomerular capillaries, a functional change in a colloid membrane of which the microscope gives no evidence. Obviously this explanation is mere surmise.

"5. Holding these views, there seems to me to be no justification for regarding nephrosis as other than a variety of kidney disease, a form of chronic nephritis (Bright's disease)."

Many others believe that "nephrosis" is simply a symptom-complex occurring in the course of subacute or chronic nephritis, in which, pathologically, degenerative changes predominate at the time.

An extremely interesting question from the clinical standpoint is whether children presenting this syndrome ever recover. Judging from the pathologic changes found in the kidneys by Wolbach and Blackfan, there seems to be no reason why they should not, if the cause is found and removed. Clausen and others believe that they have cured certain cases by the removal of foci of infection. At any rate, the urine was free from albumin two or three years later. In a condition in which remissions and exacerbations of the symptoms are so characteristic, the question at once arises, however, as to how long a child must be free from albuminuria before it can be considered cured. If albuminuria continues, even though it is slight and there are no other symptoms, it certainly cannot be considered cured. If this syndrome is simply an unusual combination of symptoms in what is commonly spoken of as subacute or chronic parenchymatous nephritis, the question arises as to whether children ever recover from this condition, even if adults do not. The probabilities are that they do not, but is it impossible that they may not from a subacute form? Whatever the final answer may be as to the prognosis in children presenting this symptom-complex, it seems to me, basing my opinion partly on the literature and partly on my own experience, that at present the outlook in such cases is very grave, although not by any means hopeless. It seems certain, at any rate, that no matter how well the child may appear, it is not safe to say that it is cured until the urine has been constantly free from albumin for several years.

Certain practical points have been learned as to the treatment, however, of children presenting this symptom-complex. In the first place, careful search should be made for a focus of infection. If it is found, it should be removed. Whether the removal of such a focus will do any good or not, is a matter of opinion. Certainly it will do no harm, and, even though the evidence that the removal of such a focus will relieve the condition is so slight, it is not fair to the patient not to seek for it and remove it, if found. It is also evident that with the low blood protein, the normal non-protein nitrogen of the blood and the normal phthalein excretion, there is no object in limiting the proteins in the food, as is usually done when the kidneys are diseased. In fact there are definite indications for giving protein freely, as it is possible that the edema may be due to the low protein content of the blood and the organism certainly needs protein to replace the loss through the kidneys. Diminution in the edema may or may not, however, follow an increase in the proteins in the food. It has also been found that a limitation of the intake of sodium chloride has no effect on the edema. Limitation of the intake of water has less influence on the edema, moreover, than in other disturbances of the kidney. It is very important to guard children with this symptom-complex against acute infections, as such infections are very likely to bring on an exacerbation of the symptoms and are often the beginning of the fatal termination. Strange to say, however, an acute infection has, in a few instances, been followed by a cessation of the symptoms and, presumably, by cure.

### "ACUTE PYELITIS" IN INFANCY

Two papers have appeared recently which seem to show that the commonly accepted ideas as to the pathological changes in this condition are erroneous. They also suggest strongly that the infection is blood-borne rather than ascending. Chown (*Archives of Disease in Childhood*, 1927, *2*, 97) reviews the literature on the subject and shows how little basis there is for the belief that there are lesions in the pelves, ureters and bladder. He reports thirty cases from the Pediatric and Pathologic Departments of the Johns Hopkins Hospital. Twenty-one cases were acute or subacute. In four of them the renal trouble was not the cause of death. He calls attention to the frequency of infection elsewhere, both enteral and parenteral. In all cases in which bacteriologic studies were made, the Bacillus Coli was found. He says that the significance of these findings cannot be determined at present. The common lesion is a suppurative, interstitial nephritis with secondary involvement of the parenchyma. The organisms and inflammatory products escape into the tubules, drain into the pelvis and escape in the urine. The pelvis is usually normal, although it sometimes appears congested. In four other acute cases in which the urine contained pus, no pathologic changes were found in the kidneys or rest of the urinary tract. In five chronic cases the pelves showed no cause for the pus, while the kidneys exhibited both acute, multiple, focal, suppurative lesions and inflammatory lesions in various stages of healing. In another instance, in which a baby died of pneumonia five months after a typical attack of "acute pyelitis", the kidneys showed nothing abnormal, proving that recovery may be complete.

Wilson and Schloss (*Amer. Jour. Diseases of Children*, 1929, *38*, 227) studied the post-mortem material from forty-nine infants and children under three years of age who had what is called "acute pyelitis." Their cases included both the type in which the chief symptoms were referable to the urinary tract and that in which the pyuria was an incident or a complication of some other acute infection. They found that the most common pathologic changes were inflammatory foci in the interstitial tissue of the kidney. In the more severe cases the changes went on to abscess formation, presenting the typical picture of acute suppurative nephritis. The milder lesions probably heal without the formation of scars. In healing, the more advanced lesions are replaced by fibrous tissue.

In twenty-eight cases the "pyelitis" was a complication of other acute infections. In only one instance was there any lesion of the pelvis of the kidney. In this case minute, focal accumulations of leucocytes were observed in the mucosa and one in the lining epithelium. There were much more marked lesions, however, in the interstitial tissue of the kidneys. Lesions were found in the kidneys in twenty-two. In one there was a mild cystitis and in five no anatomic basis for the pyuria was found. In seventeen cases, in which the pyuria and accompanying symptoms predominated, the lesions of acute, suppurative nephritis were found. In only one was there any inflammation of the pelves of the kidneys and this was insignificant in comparison with the renal lesions. In three cases that had recovered from "pyelitis" and died later from acute diseases, areas of scar tissue were found in the kidneys, but no lesions in the pelves, ureters or bladder. No lesions like those in "pyelitis" were found in the kidneys of babies that had not had pus in the urine. They admit that pyuria may be due to congenital abnormalities of the urinary tract, cystitis or renal calculi, but believe that such cases are relatively very uncommon. Their results point to a blood-borne infection rather than an ascending one. They conclude that it seems evident why the usual medical methods of treating "acute pyelitis" have been of so little value.

Helmholz (*Amer. Jour. Diseases of Children*, 1929, *38*, 968) says that the available facts as to the mode of infection can readily be used to prove it to be either ascending or hematogenous, depending on the inclination of the authors. He

states that there are undoubtedly hematogenous infections of the kidney with the staphylococcus and the streptococcus, which produce discrete abscesses in the cortex of the kidney. Whether the colon bacillus ever produces renal abscesses in the cortex of the kidney of human beings in this manner has not been definitely determined. That infection of the kidney by the colon bacillus, secondary to some stasis in the urinary tract, is common has been proved, although it is still disputed whether the colon bacillus passes to the kidney by way of the blood stream, by an ascension of the urinary passages in the lumen or through the lymphoid passages. He says that while Chown and Wilson and Schloss have shown that focal lesions in the renal parenchyma are present quite regularly in so-called pyelitis, the cases have not been studied culturally sufficiently accurately to determine the etiologic bacterial factor.

He approaches the subject from the point of view of spontaneous bacilluria and pyelitis in the rabbit. When rabbits were found to have bacilluria the ureters were tied off and the urine from the bladder and renal pelves obtained separately. He also studied the bacterial content of the urine from the bladder and ureters after intravenous injection of colon bacilli. His summary is that bacilluria in rabbits is usually limited to the bladder, but occasionally involves also the pelvis of the kidney. Exceptionally, cystitis, pyelitis and pyelonephritis are found in the rabbit. The frequency of bacilluria limited to the bladder and the relative infrequency of infection of the upper urinary passages, make it probable that the exceptional bacilluria of the upper urinary tract and pyelonephritis are the result of an ascending infection from the bladder. So far as this study of spontaneous urinary infection is applicable to conditions in human beings, the conclusion seems warranted that infections of the urinary tract in infants take place by the ascending route.

### HYPOGLYCEMIA, CONVULSIONS AND RECURRENT VOMITING

Josephs ("Fasting as a Cause of Convulsions," *American Journal Diseases of Children,* 1926, *31,* 169) in 1926 called attention to the frequency with which certain children have convulsions at the onset of any acute illness, which are proved by their cessation as the children grow older not to be due to any organic cerebral lesions. He also noted the fact that these convulsions occurred most often early in the morning before breakfast or when the child had not eaten for some hours or had vomited. In a number of cases of this sort a low blood-sugar was found at the time of the attack. It was evident from the variation in the amount of sugar found, however, that there was no definite level at which convulsions would occur in all cases, but that the level, if there was one, varied in different children. In these children the fall in blood-sugar concentration, when fasting, was more rapid and the final concentration lower than in normal children.

In the same year Josephs also wrote concerning the association of hypoglycemia and recurrent vomiting ("Recurrent Vomiting," *American Journal Diseases of Children,* 1926, *31,* 657), and quotes the meager literature on the subject. As his material was all ward patients, he was able to study only severe cases. There was a considerable reduction of the blood-sugar in the five cases studied at the height of the attack, although the blood-sugar in all was within normal limits between attacks. In all of them it was below 0.070%, which is the minimum which he found in controls as the result of long fasting. He studied a series of twenty-eight normal children, taking the blood after four and sixteen hours of fasting. In no instance was the reduction in blood-sugar greater than 12 mgs. per 100 c.cm. Age had no influence on the rate of fall. He also studied ten children, subject to recurrent vomiting, between attacks, in the same way. In all of them the concentration of blood-sugar fell more than in the normal controls. Acetone was also present in the expired air after sixteen hours in nearly all, but was never found in the controls.

The children who were studied in more than one attack showed some, but a varying, hypoglycemia in all of them. Other children, however, with similar clinical histories, had no lowering of the blood-sugar and some had hyperglycemia. It cannot be said, therefore, that hypoglycemia is the cause of recurrent vomiting in general. There are two possible explanations for the fact that the blood-sugar falls more rapidly in children subject to recurrent vomiting than in normal children; an insufficient store of glycogen or that the supply of blood-sugar is temporarily unable to meet the demand, that is, it is not sufficiently mobilized or it is used up too rapidly. He thinks the latter the more reasonable. Anesthesia, fever, overexertion or excitement, combined with a lessened intake of food, might, in a child with such a peculiarity of carbohydrate metabolism, cause hypoglycemia and thus precipitate an attack.

In 1929 Josephs wrote another paper, based on the same cases discussed in his previous papers with four additional, entitled "Spontaneous Hypoglycemia in Childhood" (*American Journal Diseases of Children,* 1929, *38,* 746). In the majority of cases an acute infection preceded the attacks. In only two instances did fasting alone seem to be the only cause. He cites the fall of blood-sugar in Marathon runners in proof that hard play may be a cause of hypoglycemia, and also his studies of blood-sugar in anesthesia, which showed a rise for a few hours and then a fall. He recommends dextrose in treatment. He thinks that repeated small doses are better than a single large dose. To prevent attacks in susceptible children avoid a long fast, even of all night. Give orange juice or dextrose

at once, if any infection. Give dextrose before anesthesia or operation. Marked fatty change in the liver was the most marked pathological change in the fatal cases. This, however, is merely the evidence of profound intoxication of some sort. The fatty change is probably secondary not primary. He calls attention to the similarity between the picture of hypoglycemia with attacks in these patients and the symptom-complex spoken of as "recurrent vomiting". He speaks of the lower value of the blood-sugar in infancy than later and the increased lability at this age. He believes that in certain instances this lability persists into childhood and may explain the constitutional factor in the hypoglycemic syndrome.

Josephs has undoubtedly called attention to an important factor in the etiology of convulsions and "recurrent vomiting" in early life. Clinical experience in many places has corroborated his opinion. He does not claim, however, that all cases of convulsions, not due to evident organic disease of the brain or meninges, or all cases of severe vomiting, are due to hypoglycemia. This, however, is what many of the younger practitioners and enthusiasts, both young and old, seem to think. At any rate, if the physician is able to give dextrose intravenously, the patient gets some, whenever it vomits or has a convulsion. Perhaps it may be safer that way, as it may do good, and probably will not do harm, unless it prevents carrying out the good old-fashioned procedures of enemata and castor oil.

Inspired by Josephs' paper, Dr. J. P. Crozer Griffith studied a number of cases which he reports under the title of "Hypoglycemia and the Convulsions of Early Life" (*J. A. M. A.*, 1929, *93*, 1526). He reports two cases of convulsions in children associated with hypoglycemia and relieved by dextrose intravenously. In one of these patients epinephrin restored him to consciousness on two occasions. The blood-sugar was raised presumably by the mobilization of glycogen by this drug. He reports two cases with acute infections in which the blood-sugar, without fasting, was low, but in which there were no convulsions. He also reports three cases of convulsions in children, manifestly due to organic cerebral lesions, in which the blood-sugar was low at the time of convulsions.

He says that it does seem to be true that there appears to be a decided tendency for convulsions to develop in the early morning before breakfast, yet in one of his cases, that had had many convulsions before breakfast, a convulsion occurred three hours after breakfast and the blood-sugar was very low. In two other cases also convulsions occurred after meals and the blood-sugar was low. He notes that "every child wakes in the morning after the night's short fast; that if the fasting *per se* produces a low blood-sugar, then every child should have hypoglycemia at this time of day, and that if hypoglycemia is the cause of the convulsions, then every child should exhibit this on waking. It is obvious, since none of these things occur, that some other factors unknown at present are at work either to permit the fasting to produce the hypoglycemia or to cause the hypoglycemia to produce the convulsions, if, indeed, there is any etiologic relationship." He notes that it is very common for a child to develop a slight digestive disturbance with fever, loss of appetite and perhaps vomiting. He may go to bed in this condition and awake with fever persisting and possibly a continuance of vomiting. The occurrence of a convulsion in combination with this syndrome is repeatedly witnessed by every physician. If this occurrence is compared with the number of cases which exhibit the other symptoms, but no convulsions, the relationship is distinctly common. It is evident again that even the combination of short starvation with fever needs something more to produce the convulsive attacks.

His studies justify Josephs' opinion that there does not appear to be any fixed level at which convulsions in children may be expected to occur, even in the same individual; and this is still more true of children as a class. He then discusses the therapeutic tests. In one case, in which the convulsions were surely epileptic, although the blood-sugar was only 38 mgs., the intravenous injection of dextrose had no immediate effect on the symptoms. This was to be expected, as Lennox and Cobb have shown that, as a rule, there is no connection between hypoglycemia and the occurrence of epileptic attacks. The almost instant recovery, on several occasions, in one of his cases and also in another, both with low blood-sugar, seems to show that the hypoglycemia was the cause in these cases. He concludes that in convulsions in children there is probably a frequent association between this disorder and the existence of a low blood-sugar content. Whether, however, this is an etiologic relationship cannot be determined from these investigations; and they show, further, that a low blood-sugar may exist without the occurrence of convulsions. In some instances at least, the conclusion is justified that a causative relationship of hypoglycemia to convulsions may be reasonably assumed.

Dr. Griffith's conclusions seem to be sane and conservative. It will be well if the enthusiasts bear them in mind.

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

## CASE 16111

## CHRONIC PANSINUSITIS AND MASTOIDITIS AND MAJOR SEQUELAE

### MEDICAL DEPARTMENT

An American-born kitchen man thirty-two years old entered from the Emergency Ward on September 20 complaining of indigestion and headache of nine days' duration.

Records of the Eye and Ear Infirmary show that he entered June 3, three months and a half before entering the Massachusetts General Hospital, complaining of obstruction of the nose for years, especially on the left side. Examination showed deviated septum. X-ray examination showed beginning destruction of the right mastoid and marked thickening of the left. The right antrum was curetted out and a large opening made into the nose. Both eardrums were perforated, with scanty discharge from the right ear. The urine showed a trace to a high trace of albumin at this time, specific gravity 1.018 to 1.024. July 12 he was discharged with a diagnosis of right maxillary sinusitis and mastoiditis, bilateral chronic sinusitis and erysipelas. At a second admission August 28 two more operations under local anesthesia were done, resection of the cartilage and bone of the septum and left ethmoidectomy and opening of the intranasal antrum. At this time two of three specimens of urine showed a trace to a faint trace of albumin. Beginning September 11 he had dull steady headache, nausea, anorexia and intermittent vomiting, persisting until admission to the Massachusetts General Hospital. A diagnosis of chronic pansinusitis was made. September 15 he was discharged from the Infirmary.

Since leaving the Infirmary his face had been puffy and dull frontal headache had persisted. On September 18 he noticed smoky urine. For two nights before entering the Massachusetts General Hospital he had been sleepless and twitchy. He had constant sour eructations and alternate constipation and diarrhea, chiefly the former. He insisted that for the past two weeks he had had no marked oliguria. He had however felt very thirsty. September 19 the dull headache became splitting and brought him to the Emergency Ward.

His family history is unimportant.

His past history is negative except that for years he had been a mouth breather and had had frequent headaches.

Clinical examination showed a fat man with an edematous face, resting his forehead on his hand, breathing 75 per cent through the mouth and 25 per cent noisily through a partially obstructed nose. Tonsils large and puffy. Location of the apex impulse of the heart not recorded. Left border of dullness 9.5 centimeters to the left, 1.5 centimeters outside the midclavicular line. Right border and supracardiac dullness not made out. No other abnormalities recorded. Blood pressure 185/120 at admission to 90/60 the day of death. Lungs clear and resonant. Upper abdomen showed slight tenderness and voluntary spasm throughout. Pupils normal. Knee jerks hyperactive.

Urine: 30 to 65 ounces, specific gravity 1.006 to 1.018. A two-hour test showed the specific gravity 1.006 to 1.012, + total output, day 625 c.c., night 900 c.c. A very large trace to a trace of albumin at all of eleven examinations. Three specimens of sediment showed occasional to many leukocytes, three others loaded with leukocytes, occasional to many red cells at seven tests, coarse granular and waxy casts at six of nine examinations. Renal function 15 to 30 per cent. Blood: hemoglobin 60 to 70 per cent, reds 3,240,000 to 3,670,000, achromia and increased platelets, leukocytes 22,900 to 16,500, polynuclears 90 per cent. Hinton negative. Non-protein nitrogen 87 at admission, 71 September 26, 115 September 28, 73 October 7 and 100 October 8. Creatinin 6 to 2. $CO_2$ combining power 57 to 33.7 volumes per cent.

Temperature 97.4° to 101°. Pulse 80 to 129. Respirations 20 to 35.

X-ray examination showed all the sinuses dull, the dullness simulating retained secretion; probably pansinusitis.

On September 22 the face was swollen, red and tender under the right eye. An eye consultant advised a nose and throat consultation. One throat consultant thought there was an acute sinus. Another thought the appearance suggested erysipelas. A skin consultant thought there was not a definite erysipelas, although there was evidently infection in the skin. A third throat consultant thought there was evidently an acute right ethmoiditis, and advised transfer to the Eye and Ear Infirmary. September 25 the patient was transferred to the Infirmary. It was thought there that there was no indication for surgical intervention. There seemed to be no pus in the right ethmoidal cells to account for the swelling. The patient was given hot saline nasal irrigations every three hours and transferred back to the medical wards the following day with the eye condition and the general condition much improved. By September 29 he showed no edema except a slight trace in the face. He continued to show distinct though slight improvement until October 5.

Then he began to go steadily downhill. He became extremely toxic, with much twitching and with signs of pericardial effusion. October 6 a pericardial tap was done in the fourth interspace 1.5 centimeters outside the nipple line. 25 cubic centimeters of blood-tinged fluid was withdrawn, not under positive pressure and coming through the needle at only one phase of the cardiac cycle. October 7 another tap in the fifth space just to the right of the sternum gave 175 cubic centimeters of grossly bloody fluid, 1,100,000 red cells. The needle was removed before all the fluid was withdrawn. It flowed freely and was not ventricular. The tap was followed by relief of the dyspnea. October 8 the patient died.

## Clinical Discussion

BY A. V. BOCK, M.D., AND GEORGE W. HOLMES, M.D.

### NOTES ON THE HISTORY

Dr. Bock: The history is very deficient. Very few men of this type would enter the Eye and Ear Infirmary with merely the complaint of "obstruction of the nose." In view of the examination it does not seem that the man would have all this pathology with the sole symptom of obstruction of the nose. He must have been fairly sick at one time or another.

Erysipelas of course is a fairly common complication of this general type of infection.

### NOTES ON THE PHYSICAL EXAMINATION

"Tonsils large and puffy." That interests me, that and the fact that the man had all this evidence of mastoid, antrum and sinus infection and trouble with both ears, and yet these tonsils were allowed to remain in place,—probably the original focus of all his trouble and the kind of thing we should want removed in dealing with the problem he presented when he first came to the hospital in June.

I think that the two-hour test is on the whole one of the best tests of the kidney function that we have. When that finding occurs—gravity 1.006 to 1.012—the diagnosis of chronic nephritis is practically made. The limit of normal output during the night should not exceed 600. The increase indicates failure of the kidney to concentrate and is a very good index of nephritis. If it were not for the fact that he has had sepsis for months I think an anemia of 3,200,000 would go perfectly well with chronic nephritis that had advanced toward a terminal stage. Very few cases of chronic nephritis come to death without developing an anemia. The increased blood platelets are very characteristic of the type of infection that he has. Any subacute or chronic infection may cause an increased output of blood platelets. His blood count of course is unusually high if one takes into account only the infection in the head. If the history is correct that he passed smoky urine before he came in—and the microscopic picture suggests that it is correct, because they found blood in the urine—it suggests that he has an exacerbation of chronic nephritis.

Erysipelas is sometimes a very difficult diagnosis to make. We see erythema of the skin sometimes with sharply demarcated margins, and often it is a question whether we are dealing with erysipelas or cellulitis not due to real erysipelas infection.

Terminal pericarditis does not help very much in the underlying diagnosis. We know that pericarditis occurs as a terminal incident in a great many conditions. We know it is a common terminal finding in nephritis, and in this case it appeals to me as being a part of the terminal picture of a chronic nephritis.

### DIFFERENTIAL DIAGNOSIS

The origin of his nephritis and how long he has had nephritis we cannot say. The amount of blood and white cells and casts in the urine suggests an acute nephritis. I think there is some stirring up of an older nephritic process in these kidneys. He has had enlargement of the heart, high blood pressure, low specific gravity, all earmarks of chronic nephritis. In this case in a man of thirty-two we do not expect this to be the arteriosclerotic type of nephritis with the small granular type of kidney but presumably a kidney of fairly normal size with the process in the glomeruli, although we have made predictions often enough on the wards to find that all sorts of guesses may be wrong at autopsy. I should say that the small arteriosclerotic type of kidney does not occur at thirty-two years of age except as the end result of prolonged bacterial endocarditis. In this patient at this age I should say that while all the rest of the picture—the high blood pressure and the retention of nitrogen and the fixation of specific gravity—are like that type of nephritis, it would be unusual to see it. He had a rather widespread infection. It is difficult to say what the origin of that infection was, but it might very well have started from the tonsils.

Dr. George W. Holmes: This film was taken for study of the mastoids. The dark round spots are the bony canal and these wing-like shadows on each side are the mastoids. The film is taken so that we have the opportunity to compare one mastoid with the other. The white ring around the canal is normal. That is the dense part of the mastoid. Beyond that you see bubble-like shadows that represent mastoid cells. Dr. MacMillan interpreted this film as disease of the mastoid on both sides, and I would not attempt to change his interpretation. He sees a great many more of these things than I do. The points we look for are thickening of the wall and general haziness in appearance, and later on complete obliteration of cells.

These films are taken to show the nasal and

frontal sinuses. The dark areas on each side are the frontal sinuses. In this region are the ethmoidal and sphenoidal sinuses. It is quite obvious that the antra are dull. The ethmoidal and sphenoidal are also dull. I think there might be a question as to whether the frontals are normal or not. The difference in density is slight, and of course that varies with the depth of the sinuses. From this film you can get some idea of the depth of the sinus and the thickness of the wall in front of it. If you have a heavy anterior wall and a shallow sinus, the shadows are more dense than in the reverse of this condition. This patient has a rather shallow sinus and heavy anterior wall and one might say they were normal. Another point of some value is sharpness of the edge of the sinus. This seems to me to be fairly sharp. There is no doubt of disease of the antrum, probably of the ethmoid region, and questionable sinus disease.

This chest film was taken on June 5. The lung fields are about what we should expect. There might be a question of some thickening in the upper part of the left lobe, but that is very slight and I do not believe it has anything to do with this clinical trouble. The apex is a little dull. You might find some scarring of the apex and probably some thickening of the pleura on that side. I should not expect an extensive involvement in the lung. There is nothing abnormal in the size of the heart and the appearance of the great vessels, although that film is not taken for the heart. It certainly does not give the impression of a large heart; and if he has had a long-standing nephritis with high blood pressure we should expect some change in the heart. The change in the upper portion of the left lung is rather more noticeable in this later film. It is not likely there would be such a change in a patient in that period of time. It is probably due to a difference in the films. This was probably taken with a little more penetrating light than the previous one. While the heart shadow is a little round and there is prominence of the curve of the left ventricle, we have very little on which to base a statement that the heart is enlarged.

Dr. Bock: The problem of the cause of the nephritis of course is interesting. We see nephritis complicating scarlet fever. Nephritis is often associated with acute infections. We get a history oftentimes of acute nephritis following severe tonsillitis. That is not very uncommon in children, I think more common than it is in adults. The question in this case is whether the damage to the kidneys has had its origin in the sepsis throughout the sinuses and antra or whether it got started some time before that, possibly through the tonsils. I do not think we can answer that. One is inclined to tie the two together, especially in view of the fact that the X-ray findings do not substantiate the finding of a very big heart and the physical examination did not show that the man had anything like the maximal hypertrophy we see in nephritis of long standing with such urine findings.

I should say considering the age of the patient and the infections in the sinus, antrum and mastoid that the patient died of chronic glomerular nephritis.

A Student: Was the blood from the pericardial fluid traumatic?

Dr. Bock: It is hard to say. I think there was more blood there than in straight pericardial fluid. It probably was traumatic.

### Clinical diagnoses (from hospital record)

Chronic nephritis.
Pansinusitis.
Uremia.

### Dr. A. V. Bock's diagnoses

Chronic glomerular nephritis.
Infections in the sinuses, antra and mastoids.
Terminal pericarditis.

### Anatomic diagnoses

1. *Primary disease.*

Subacute glomerular nephritis.

2. *Secondary or terminal lesions.*

Terminal pericarditis.
Hypertrophy of the heart.
Completely organized lobar pneumonia, upper lobes of both lungs.

### Pathologic Discussion

BY TRACY B. MALLORY, M.D.

We did find nephritis at autopsy. The kidneys were slightly above normal in size, 360 grams. In gross the capsules stripped easily, but on the pale surface were many hemorrhagic spots. The gross picture is that of subacute nephritis of a few months' to a year's duration. There was a terminal pericarditis. The heart was considerably hypertrophied, 480 grams.

The microscopic examination showed a marked glomerular lesion, with proliferation of endothelial cells and occlusion of the capillaries. There was practically no atrophy of the tubules such as occurs in the chronic stage, but we found a slight unquestionably recent acute tubular process as well as the glomerular one. I should certainly guess from the history that the entire renal involvement was less than six months old. We have a history of sepsis of not more than four months, and I think the kidney lesion started about simultaneously. If this is so we must assume that the heart hypertrophied up to 480 grams in four months, for that is the period between the time of the X-ray film Dr. Holmes showed us and the time of autopsy. I know of no figures on the question of the rapidity of cardiac hypertrophy, so that the case is quite interesting from that point of view.

There was another autopsy finding of considerable interest which was impossible to diagnose

on the face of the records, and that was a completely organized lobar pneumonia of both upper lobes. It is rather striking that there should have been no physical signs to show for it at the time he was in the hospital here. The infectious process was all over at the time of autopsy and his lungs was pretty well fibrosed. Of course something would have shown if he had had a second X-ray at this time but apparently nothing was found to suggest taking one. The so-called unresolved pneumonia, a diagnosis I was taught to avoid like poison in medical school, since it so commonly proves to be empyema, nevertheless does occur. This is about the fifth or sixth case of organized pneumonia we have had in a year.

DR. HOLMES: Was there anything at autopsy to account for the retracted apex?

DR. MALLORY: No. I did not find anything that helps.

DR. BOCK: It is interesting that kidneys which showed what these did should have put out urine showing so much fixation of specific gravity, characteristic of old scarred kidneys.

## CASE 16112

## A CASE OF CHRONIC URINARY INFECTION

### UROLOGICAL DEPARTMENT

An American carpenter thirty-three years old entered May 1 complaining of frequency and burning on micturition of five or six years' duration.

Thirteen years before admission he had catheterization following appendectomy. The catheter was passed six or seven times. Urinary disturbances began at that time and had persisted ever since with increasing severity. Five years before admission he was treated for stricture of the urethra. He had frequency and burning micturition. A cystoscopic report from the hospital where he was seen at that time states: "The left ureter was catheterized and urine containing a few leukocytes and red cells withdrawn. No tubercle bacilli were found. Function 10 per cent in half an hour. The right ureteral orifice was said to be sclerosed. It could not be catheterized." He was operated upon for diverticulum of the bladder. Bladder irrigations were attempted but had to be given up because the pain was so intense that he could not endure it. The frequency continued. February 24, a year and three months before admission, he came to the Out-Patient Department of this hospital. The urine showed the slightest possible trace of albumin, many leukocytes, 3 red cells per high power field. A culture showed streptococcus viridans. Non-protein nitrogen 31 milligrams. A guinea pig test was negative for tuberculosis. The bladder was washed. He did not return to the Out-Patient Department for a year. Then his symptoms increased, and February 6, three months before admission to the wards, he returned. The residual urine was one ounce. The right kidney was palpable. At X-ray examination the kidney outlines were not well shown. The left kidney appeared larger than normal. There was an irregular calcified mass overlying the sacrum, probably a calcified gland. Cystoscopy showed the bladder mucosa red and generally inflamed, with much exudate over the base. No diverticula were seen. Both ureteral orifices were hills of edema. The catheter was passed up the right ureter for 15 centimeters with difficulty. The catheter was engaged in the left ureter but could not be passed. In the Male Medical Department no tuberculosis of the lungs was found. A diagnosis of chronic bronchitis was made. February 19 a pyelogram showed that the injected right ureter and kidney pelvis were abnormal. The major calices were elongated and narrow. The usual cupping of the minor calices was normal. The ureter appeared dilated and tortuous. Several areas of constriction were possibly present in the ureter. The left kidney appeared larger than the right. The pelvis of the right kidney was small. The dense irregular calcified mass overlying the sacrum was still present though it had shifted its position.

In the wards the patient gave a history of pain beginning at the end of the penis every forty minutes. It took some time to start the flow of urine. As soon as he passed 25 to 100 cubic centimeters the pain was relieved. Pyuria and albuminuria had been constant. His physician had been treating him with urotropin. Recently when he strained to urinate he had a few small bowel movements. His frequency was now fifteen times by day and eight to ten times by night. He had lost no weight.

His family history is not significant.

He had a peritonsillar abscess when young. Nineteen years before admission he had tonsillectomy and submucous resection. Eleven years before admission he was gassed in France. The following year he had glands in the left side of the neck persisting for two months. About a year later he began having "asthmatic attacks" with cough and frequent sputum. These persisted until admission, though he had had only two in the past year. He was rather susceptible to "colds," and when he had them was quite ill, with much cough and sputum and slight dyspnea.

Clinical examination showed a thin young man with poor muscles. On expiration constant musical râles were heard in both lungs anteriorly. The lungs showed normal resonance. Heart sounds very distant. Apex impulse not seen or felt. No enlargement to percussion. No murmurs or other abnormalities. Pulses and arteries normal. Blood pressure 120/88. Slight costovertebral tenderness in the left loin. Abdominal wall relaxed. A median suprapubic scar extending to two inches below the umbilicus. There was a definite hiatus in the muscle wall in this scar. Right McBurney's scar.

Before cystoscopy urine cloudy, specific gravity 1.014, a very slight trace of albumin. Sediment, 15 leukocytes per high power field. Renal function in two hours 80 per cent. Non-protein nitrogen 31 milligrams. Hinton negative.

May 3 cystoscopy was done. The observation cystoscope was passed with some difficulty to the bladder. Instrumentation was quite painful. The bladder was very intolerant. Except for a considerable degree of cystitis nothing definite was made out. The function for the right ureter was 5 per cent, for the left 3 per cent. Urine from right ureter showed many red cells, no leukocytes. That from the left showed a moderate number of red cells, 5 to 10 leukocytes per field, with a few clumps. Cultures from both urines were sterile.

The patient was put upon constant drainage. There was a little bleeding after the cystoscopy stopped by hot irrigations of boric with adrenalin. He ran a little temperature and had soreness in the costovertebral angles. May 7 pyeloli* was started. May 8 the patient was better and May 10 was up in a chair with the temperature down.

May 13 a second cystoscopy was done.

## Clinical Discussion

BY GEORGE GILBERT SMITH, M.D.

The history up to this point is that of a long-continued urinary infection which apparently was started by catheterization, and was kept up by a bladder diverticulum. The removal of this diverticulum did not clear up the infection. As so often happens in cases of this sort, the bladder neck became sclerotic and contracted. Tuberculosis was ruled out by guinea pig test. The infection involved both kidneys, and the ureters showed considerable dilatation. This dilatation was probably due to two factors, (1) obstruction at the vesical wall caused by the frequent spastic contractions of a hypertrophied bladder, and (2) replacement of the musculature of the ureter by fibrous non-contractile tissue. This local condition was found in a thin, asthenic young man whose powers of resistance had been much diminished.

## Further History

The second cystoscopy was done under a small dose of spinal anesthesia with entire absence of discomfort to the patient. The bladder urine was turbid. The bladder was generally reddened. There was a good deal of pus on the base. No diverticular orifice could be seen. Just above the trigone there was an irregular round area which was either neoplasm or a large collection of pus and mucus. Bleeding very quickly obscured the view and it was impossible adequately to examine the bladder. Both ureteral orifices were seen and except for the surrounding inflammation did not seem remarkable. Catheters passed in both sides. A little difficulty was found in passing the catheter the full distance to the kidney.

Bilateral pyelograms showed the kidney outline apparently normal. There was an anatomic variation in each kidney pelvis characterized by an unusually long upper calyx ending in a rudimentary pelvis with several minor calices branching out from it. The right pelvis and ureter showed about the same dilatation as before described. There was also moderate dilatation of the left ureter. The calcified masses previously described over the sacrum were still present.

May 15 operation was done.

## Further Discussion

The object of the operation was to provide opportunity for thorough examination of the bladder and to substitute suprapubic drainage for the obviously ineffectual urethral drainage.

### PRE-OPERATIVE DIAGNOSIS

Chronic cystitis.

### OPERATION

Ether. The bladder was opened and thoroughly inspected. The wall was moderately thickened, but the size of the bladder appeared normal. The mucosa was generally smooth and pale. In the dome was a cluster of small wormy ulcerations corresponding with the area which had been seen through the cystoscope. Both ureters appeared rather tight, so the orifices were cut back with a diathermy knife. The bladder outline was found to be very tight and fibrous. A good sized segment was removed with the diathermy knife, thus opening the orifice. The bladder was closed about a tube.

### PATHOLOGIC REPORT

Chronic inflammation.

## Further Discussion

A localized ulcerative cystitis was found which was thought to account for the patient's bladder symptoms. The cause of the ulceration was thought to be the intense infection which was kept up by urinary retention in the dilated ureters and by the tight bladder outlet. All that could be done to remedy these conditions was done.

## Further History

The patient was troubled with his asthmatic condition following the operation. Benzoin inhalations helped this. The wound healed slowly. May 25 the tube came out and was replaced by a mushroom catheter. June 9 he was discharged.

*History of interval.* For the next five months he felt fairly well. Early in November he had the suprapubic tube removed and the supra-

*A Danish preparation, tablets containing 0.6 gm. calcium chloride with a coating of about 0.3 gm. of phenyl salicylate. Intended as a urinary antiseptic.

pubic fistula promptly closed. After that he had frequency increasing until he urinated nearly every ten minutes morning and night, with extreme burning on micturition, made worse when he tried to retain his urine. After the removal of the tube he had specks of blood in the urine. Beginning November 24 the urine showed gross blood. Throughout the six months he had his old cough, relieved somewhat by smoking a special brand of cigarettes. He gained weight during the first five months and a half, increasing from 115 pounds to 135. During the last two weeks of the interval he dropped back to 116.

*Second admission.* December 2, six months after his discharge, he reëntered.

On clinical examination he was emaciated, breathing hard and complaining of burning around a catheter which had been fastened in the urethra. Skin dry and pale. Small shotty cervical and axillary glands. Tongue and throat red and dry. Chest somewhat sunken in at lower rib margins in front. Ribs very prominent. Lungs full of crackling and squeaking râles. Heart sounds of fair quality; hard to hear because of râles. Apex impulse not recorded. Left border of dullness 9 centimeters to the left of midsternum, .5 centimeter outside the midclavicular line. Right border at the sternum. Supracardiac dullness not recorded. A systolic murmur. Artery walls palpable and tortuous. Blood pressure 130/80. Abdomen tense, not easily palpated. Broad median scar in the lower abdomen with a very thin covering over the intestines,—a potential hernia. Scar of the appendix operation on the right. Extreme tenderness at the rectal orifice and throughout the rectum. Shins sharp. Pupils equal, regular, reacted sluggishly to light and distance. Knee jerks normal.

Amount of urine 43 to 188 ounces daily, usually 60 ounces or more. Before operation urine alkaline at all of four examinations, cloudy at three, red at one, specific gravity 1.006 to 1.012, a slight trace to a large trace of albumin at all examinations, sediment loaded with red cells once, 10 to 15 per high power field at another. Blood: hemoglobin 70 per cent, reds 3,870,000. Non-protein nitrogen 43.

Temperature elevated to December 18, ranging from 99.5° to 102.6° daily, normal to December 22, then 96.1° to 100° until operation. Pulse before operation 160 to 72. Respiration slightly rapid for the first week, to 32, afterwards normal except for increase to 40 December 17 after transfusion.

X-ray examination. The lumbar spine and pelvis showed no definite evidence of metastases. Overlying the sacrum there was an irregular area of calcification possibly due to calcified glands or possibly foreign material in the bowel. The film showed slight motion.

December 7 a heart consultant reported: "This patient seems to me to be in very poor general condition. He looks the picture of chronic sepsis, presumably of the lungs and bladder. He is a problem for a chronic hospital. The cardiovascular system seems negative. If operation is done I would suggest transfusion. Get blood examination."

The patient was very uncomfortable at first, with constant burning in the urethra. He was put upon constant drainage. By December 8 he was more comfortable. Carious teeth were extracted. December 16, 300 cubic centimeters of blood were transfused. December 23 the catheter was removed but had to be replaced two days later because of strangury. He made good improvement.

January 5 operation was done.

### Further Discussion

This course was decided upon only after much thought and consultation by the staff. It was believed that the patient was toxic from his renal infection, and that this infection could not be cleared up except by direct drainage of the kidneys either by nephrostomy or by ureterostomy. After fair trial of suprapubic drainage of the bladder, his condition had not been improved. It was decided therefore to do a permanent ureterostomy.

### PRE-OPERATIVE DIAGNOSES

Chronic cystitis.
Chronic pyelonephritis.

### OPERATION

Ether and spinal novocain. Median suprapubic incision. The peritoneum was stripped off the right pelvic wall and the ureter found to be dilated to the diameter of 1.5 centimeters. Just before entering the bladder it ran into a mass of scar tissue two centimeters in diameter. The ureter was cut as low as possible. The lower end showed a tortuous channel and several strictures. The ureter was resected at a point above this obstruction and a soft rubber catheter passed to the kidney pelvis. The ureter was brought out through a stab wound just inside the right anterior-superior spine. It was under some tension. A similar procedure was followed with the left ureter, which was also markedly dilated and strictured at the lower end. The process was however not quite so marked as in the right. Rubber drains were placed to both sides of the pelvis and to the iliac fossae. The wound was closed carefully about these drains in an attempt to obliterate the hernia.

### PATHOLOGIC REPORT

Chronic inflammation.

### Further Discussion

The findings at operation justified the opinion previously held that the pyelonephritis was being kept up by obstruction at the lower ends of the ureters. The degree of obstruction was indeed much greater than had been expected.

### Further History

January 9 there was fair drainage from both ureterostomies. The patient's chest filled up with loose moist phlegmy râles. January 11 the right foot seemed practically paralyzed. It rotated internally. January 13 all the suprapubic stitches except the through and through stitches and all the stitches about the ureteral openings were removed. Both ureters were draining well. The cough was somewhat better.

January 19 the patient was irrational, with a pulse of 195, temperature 103.5°, respirations 30. The following day he died.

### Further Discussion

The postoperative course was what might have been expected in a patient so depleted by sepsis. There was no definite change in his condition, but merely a gradual failure. His kidneys drained well. The fact that the improvement in renal drainage did not bring about an improvement in his general condition suggested that other foci of infection were still active. There was extensive suppuration from the abdominal sinus, but this could be washed out and drainage appeared to be unobstructed. We knew the chronic pulmonary infection was still present, and thought that might be a serious drain upon his strength. The clinical causes of death were pyelonephritis, suppuration about the sinus, chronic bronchitis.

### Anatomic Diagnoses

1. *Primary disease.*

   Chronic pyelonephritis.

2. *Secondary or terminal lesions.*

   Acute endocarditis and myocarditis.
   Chronic and acute bronchiolitis.

3. *Historical landmarks.*

   Cystostomy.
   Ureterostomy, bilateral.

### Pathologic Discussion

BY TRACY B. MALLORY, M.D.

This man presented at autopsy a picture of unusually generalized sepsis, and it is difficult to know which features of it were most important. Little information was added as to the genito-urinary condition. The ureterostomy wounds were in good condition, and there was no obstruction to drainage. The ureters were dilated, their walls thickened. The pelves showed rather little evidence of involvement, but both kidneys were much enlarged and showed a diffuse interstitial infectious process with secondary destruction of the tubules and to a less extent of the glomeruli. The bladder still showed a marked cystitis.

The lungs showed an extremely extensive bronchiolar involvement. Some of the bronchioles were dilated and filled with pus to form small bronchiectatic cavities. Others showed destruction of the mucosa and ingrowth of fibrous tissue, a process suggesting bronchiolitis obliterans. There was marked interstitial fibrosis and lymphocytic infiltration of the alveolar walls throughout the lung.

The heart showed acute endocarditis and a focal myocarditis. A contamination prevented us from determining the organism. A small septic focus was found in the pancreas and numerous small abscesses and one large abscess in the spleen.

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## A CONSULTING SERVICE FOR EPIDEMIC MENINGITIS

A CONSULTING diagnostic service for epidemic meningitis under the auspices of the Department of Public Health and comparable with that now offered for poliomyelitis by the Harvard Infantile Paralysis Commission marks a forward step in communicable disease control. Upon request of the attending physician, a representative of the Department will see a case of suspected meningitis with him, and if so desired will be ready to confirm the diagnosis by laboratory methods at the bedside.

Every communicable disease presents special problems which must be met in attempting control. For diphtheria the State now offers free diagnostic laboratory service; this same service for meningitis must be available at the bedside. Attempts to check the spread of the disease have been notoriously discouraging but early diagnosis and treatment have materially contributed to a lowered fatality. It is hoped that through the proposed service diagnostic facilities comparable to those found in large hospitals may be made readily available to every physician within the State.

While the meningitis situation in Massachusetts is not alarming, the increasing prevalence throughout the country should prompt every practitioner to watch for the disease. The establishment of a meningitis service is a valuable precaution against a future emergency. Its success will depend upon the reception accorded it by the profession.

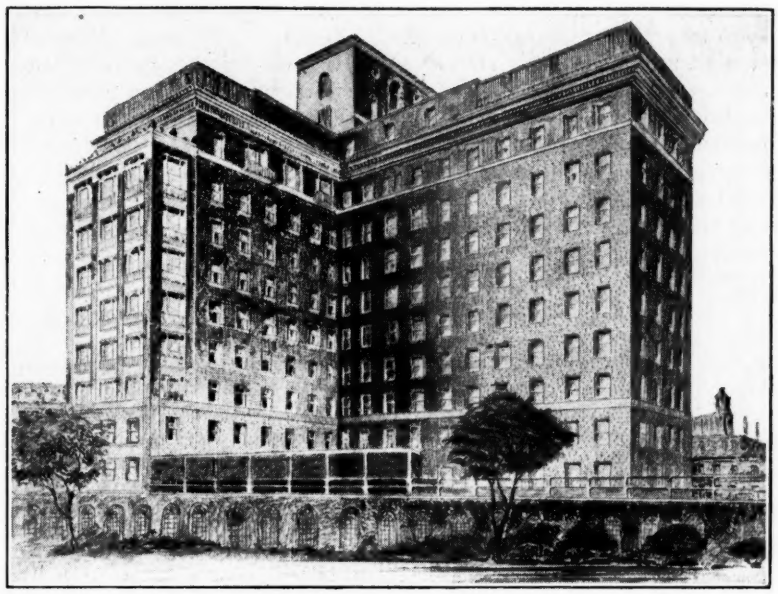
## DEDICATION OF THE BAKER MEMORIAL HOSPITAL

THE new Baker Memorial Hospital for people of moderate means was dedicated at the Massachusetts General Hospital on February 27, and the first patients were received on March 3. The dedicatory exercises took place in the rotunda of the Moseley Building, being opened with a prayer by Rev. Henry K. Sherill, rector of Trinity Church and a trustee of the hospital.

Addresses were made by Governor Frank G. Allen who lauded this new departure, and Edwin R. Embree, of Chicago, president of the Julius Rosenwald Fund, which contributed to the building of the hospital and has offered to defray half the deficit for the first three years. Mr. Embree pointed out in his address that, while under conditions of poverty and dependence, charity was a virtue and a human necessity, today the citizen of America does not require alms nor want charity. He wants and demands that in medical treatment, as in other necessities of life, agencies be so organized that service will be good and efficient and costs correspondingly low. Under new conditions, Mr. Embree said, hospitals should take increasing pride in striving to have all their services so organized that each patient, even the low wage-earner, may pay as he goes for what he gets.

George Wigglesworth, president of the hospital corporation, and William Endicott, chairman of the board of trustees, described the years of planning which preceded the construction of the building, beginning with the idea started in 1914 by Dr. Frederic A. Washburn, director of the Massachusetts General Hospital, and praised the generosity of Mrs. Mary Rich Richardson, who bequeathed $1,000,000 towards the building in memory of her father, Richard Baker, Jr., and her mother, Ellen Maria Baker. After the exercises the building was inspected by the large number of guests, and tea was served.

The first floor contains offices and the x-ray department, the second a clinical laboratory, a school for technicians and some rooms for patients. The ninth floor is for obstetrical cases, and the eleventh for operating rooms. The building will eventually contain 333 beds, of which 189 are now available. Room charges vary from $4 a day for a bed in a nine-bed room to $6.50 a day for a single room. In a four-



BAKER MEMORIAL HOSPITAL.

bed room the cost is $4.50 a day, and in a two-bed room $5.50 a day. Special medical and surgical fees will be arranged with a maximum of $150. The hospital will be staffed entirely by the staffs of the Massachusetts General Hospital and the Massachusetts Eye and Ear Infirmary with Dr. W. Franklin Wood as superintendent.

## MEDICAL ETHICS: WHAT IS IT?

WHATEVER else medical ethics may prove to be on careful investigation it is not a system of rules of conduct which, based on the experience of the past, is so formulated that it will apply exactly to every occasion in the future. "New occasions teach new duties".

In rejecting something which has come to us by tradition and in appropriating something that the insistent present offers, selection is necessary. Not everything from the past is to be thrown away, not everything that the present urges upon us is to be called good. In accordance with what principle or principles shall we select?

In seeking for a criterion of what the physician ought to do, which may help in answering also the more difficult question of what the physician may do, it is important to recognize the difference between a profession and that which is not a profession. The contrast suggests several antitheses. There is the professional and the amateur. Clearly this is not the distinction we are seeking. There is the professional and the commercial. This is a closer approximation but one notes the development of the point of view that business also may be a profession. There is the professional and the trader. This brings us very close to what we desire, the distinction between a profession and a trade.

The idea of a profession involves the idea of *noblesse oblige*. There is a certain obligation involved, a certain responsibility in medicine, arising out of what the physician is, and what the patient is and what their relations are, namely, a relational responsibility.

In trade there were originally no obligations. *Caveat emptor,* let the buyer beware. Trade and barter are closely allied and there has come down to us in connection with the word "barratry" (the same root as barter), a special form of deceit or cheating. In a trade it appears too often that one gets all one can and gives as little as possible. In a profession there is an additional factor, sometimes not clearly defined. It is recognized by trade and exemplified in some trade maxims. "Honesty is the best policy". Strictly speaking policy should involve ethical considerations, but too often it means absence of such implication. "Service pays". "Ministering" to the needs of others, does not yet, in the trade sense "pay".

Medical ethics comprehends the relations of the physician and the patient (and family), the physician and his fellow physicians, the physician and the larger community of which he is a part. It is based on the Golden Rule, the application of which between individuals may be a little clearer than between an individual and society.

An acute problem now, concerns advertising by a physician. To what extent is this proper? The term advertising, unless depolarized, and that is something hard for us to accomplish, has trade connotations which prejudice the reply. The correct professional attitude for the physician is to seek to be known for what he is. This is so far removed from what is commonly meant by advertising that it seems better not to use that term.

In a small community "known" and "known for what he is" soon may come to mean the same thing. Whatever a physician is, he is likely to be found out promptly in the small community. In the larger communities, as large cities, he may live for years without detection. He may become well-known through the newspapers, as often happens; and if nothing personally discreditable appears, it may give him prestige with a certain group of patients. Sometimes alleged personal discreditability is a paying advertisement with certain other groups in the community.

The physician seeks to be known for what he is by becoming a part of the community in which he makes his home. He shoulders responsibilities as husband and father, as a member of his family, of his clubs and societies, his profession, his town or city and state, his church. He may consciously employ any or all of these to help him make money. But in so far as he does this he leaves a profession and enters a trade.

The question is sometimes asked: why does not a physician advertise just as does a man in business? Of course, the answer is because he is not just as a man in business. The chances are that the "man in business" is a trader. The chances are that the physician is not a trader. He does not advertise as a trader because he is not a trader, he is a member of a profession.

The future will prove to be different from the past and the present in which we act is different from anything we have experienced before. There can be no exact formulation of rules which will cover every case that may present itself. There will always be a penumbrous field in which one person may see more clearly than another, and each one must do as *he* sees best. There is no intrinsic wrong if a physician advertises, even as a trader. But if he does it, he is no longer the professional. The guiding principle with a profession is *noblesse oblige*. If there is no *noblesse*, there is, of course, no *oblige*. Perhaps this is one of the difficulties with so many doctors today.

## THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

DEAVER, JOHN B. LL.D., Sc.D., M.D. University of Pennsylvania School of Medicine 1878. F.A.C.S. Surgeon-in-Chief, Lankenau Hospital, Philadelphia. Emeritus, John Rhea Barton. Professor of Surgery, University of Pennsylvania. Professor of Surgery, Graduate School of Medicine, University of Pennsylvania. His subject is: "Cholangitis and Hepatitis." Page 513. Address: 1830 DeLancey Place, Philadelphia, Penn.

ANDERSON, GAYLORD W. A.B., M.D. Harvard 1928. Epidemiologist, Massachusetts Department of Public Health. Address: Room 546, State House, Boston. Associated with him is:

BIGELOW, GEORGE H. A.B., M.D. Harvard 1916. Doctor of Public Health 1921; Commissioner of Public Health for Massachusetts. Address: State House, Boston. Their subject is: "Epidemic Meningitis Control." Page 518.

MCKHANN, CHARLES F. A.M., M.D. University of Cincinnati College of Medicine, 1923. Assistant Professor of Pediatrics, Harvard Medical School. His subject is: "Meningococcus Meningitis in Infants." Page 520. Address: 240 Longwood Avenue, Boston.

GILDEA, EDWIN F. A.B., M.D. Harvard 1924. Formerly Assistant in Neuropathology, Harvard Medical School. At present Research Assistant. Institute of Human Relations, Yale University. Address: 375 Park Street, New Haven, Conn. Associated with him are:.

KATTWINKEL, EGON E. A.B. Fourth Year Student, Harvard Medical School. Address: 45 Margaret Road, Newton Highlands, Mass. And

CASTLE, WILLIAM B. M.D. Harvard 1921. Assistant Professor of Medicine, Harvard Medical School. Associate Physician, Thorndike Memorial Laboratory, Boston City Hospital. Address: 218 Newbury Street, Boston. Their subject is: "Experimental Combined System Disease." Page 523.

OSGOOD, ROBERT B. A.B., M.D. Harvard 1899. F.A.C.S. John B. and Buckminster Brown Professor of Orthopedic Surgery, Harvard Medical School. Chief, Orthopedic Service, Children's Hospital. His subject is: "The Governor's Welfare Program and Certain Aspects of Chronic Disease." Page 527. Address: 372 Marlborough Street, Boston.

PECK, EUGENE C. A.B., M.D. Harvard 1919. Instructor in Physiological Chemistry, Tulane University of New Orleans, 1920-21. Assistant in Pediatrics, Harvard Medical School 1921-22. Professor of Biochemistry and Physiology, St. John's University, Shanghai, China 1922-25. Pediatrician, Lawrence and Memorial Associated Hospitals, New London, Conn. His subject is: "A Simple Therapeutic Arc." Page 529. Address: 138 Huntington Street, New London, Conn.

BRUEN, CURTIS. A.B., M.D. Cornell University Medical College 1928. His subject is: "Chart for the Computation of Harris-Benedict Standards of Basal Metabolism." Page 531. Address: Towaco, New Jersey. Professional address: New York City.

MORSE, JOHN LOVETT. A.M., M.D. Harvard 1891. Professor of Pediatrics Emeritus, Harvard Medical School. Consulting Physician at Children's Hospital, Infants' Hospital and Beth Israel Hospital. Member, New England Pediatric Society, American Pediatric Society, Association of American Physicians and Boston Obstetrical Society. His subject is: "Progress in Pediatrics." Page 533. Address: 483 Beacon Street, Boston.

## The Massachusetts Medical Society

SECTION OF OBSTETRICS AND GYNECOLOGY

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Frederick J. Lynch, M.D., *Clerk*

*Will you please discuss diabetes and pregnancy?*

The occurrence of glycosuria during pregnancy is common: the coexistence of pregnancy and true diabetes mellitus is rare, because the diabetic female of child-bearing age is notoriously unfertile. Von Noorden's statistics, often quoted, indicated that in a series of 427 diabetic women reported by him only 5% became pregnant. During the years 1916-1928 inclusive the house service of the Boston Lying-In Hospital, reporting the delivery of 15,869 patients, yielded only 22 deliveries of 20 diabetic patients.

Each pregnant diabetic presents a problem which must be handled according to the circumstances attendant upon the individual case. Whereas in pre-insulin days the occurrence of pregnancy in a severe diabetic was looked upon as a serious indication for abortion, so many such cases today can be carried safely along because of the availability of insulin that no hard and fast determination to terminate pregnancy in the diabetic patient can be considered justifiable. Each case must be handled upon its own merits.

From the standpoint of diagnosis, the diabetic patient may be differentiated from the non-diabetic according to two broad lines of classification:

1. The patient who is known to be a diabetic before the occurrence of pregnancy. Here the history, the reports of previous blood-sugar determinations and urinalysis are of value.

2. The patient who before pregnancy was not known to have diabetes, or in whom diabetes develops during gestation. In this type of case the typical symptoms of polydipsia, polyuria, and weight loss may occur in a striking way, but in their absence the diagnosis must be sought by careful and repeated urinalyses, blood-sugar determinations, and measurement of the patient's carbohydrate tolerance.

The diagnosis of coexistent diabetes and pregnancy having been made, it is rational treatment to allow pregnancy to continue at least as long as the diabetes can be controlled by diet and insulin. In this connection Joslin has written that "diabetes (in pregnancy) is amenable to treatment, but it tests one's skill. From beginning to end the patients must be watched, the urine examined weekly, bi-weekly, and daily near term. . . . Severe acidosis can appear in a few hours. . . . Treatment of diabetes in pregnancy is along the very same lines as the treatment of diabetes apart from pregnancy, simply bearing in mind the sudden transition which may occur in these individuals and the pains which must be taken to adapt the diet to their diabetic whims."

Inasmuch as the ability to carry pregnancy through to a successful termination in a diabetic patient is dependent upon (1) the severity of the diabetes, (2) the effect of advancing gestation upon the damaged maternal pancreas and correlated endocrine glands, and (3) the effectiveness of treatment, it is impossible to give other than a guarded prognosis. The neglected or untreated pregnant diabetic is exposed to the dangers of suddenly developing acidosis and coma, and of abortion, premature labor, and intrauterine death of the fetus. If conscientious antidiabetic treatment has failed to check the course of the disease, pregnancy should be terminated before viability of the child; (1) if toxemia supervenes; (2) if 30 calories per kilogram of body weight cannot be handled; (3) if hyperglycosuria or acidosis persists. After viability, labor should be induced if the above conditions obtain, plus hydramnios and progressive weakness.

Questions of a similar nature to the above will be discussed in the JOURNAL each week. They may be addressed to the Clerk of the Committee in care of the JOURNAL and will be answered by members of the Committee of the Section of Obstetrics and Gynecology.

## MISCELLANY

### MISTAKEN IDEAS ABOUT RABIES

The common impression that rabies is especially a disease of warm weather is not regarded as true by the State Board of Health of Missouri because more virulent types of this disease have been encountered in that state during winter months.

Of the ninety-three positive heads received by this department in 1929, fifty-six were from rabid dogs during the winter months.

### WEEKLY HEALTH INDEX

Telegraphic returns from 65 cities with a total population of thirty million for the week ending March 1, indicate a mortality rate of 14.7 as against a rate of 15.7 for the corresponding week of last year. The highest rate (24.4) appears for Trenton, N. J., and the lowest (6.9) for Des Moines, Iowa. The highest infant mortality rate (200) appears for Camden, N. J., and the lowest for Fall River, Mass., Schenectady, N. Y., and Tacoma, Wash., which reported no infant mortality.

The annual rate for 65 cities is 13.9 for the nine weeks of 1930, as against a rate of 17.2 for the corresponding weeks of 1929.

## RECENT DEATH

**McCLURE**—DR. WILLIAM LIONEL MCCLURE, a well-trained and popular young surgeon of Boston, died at the Massachusetts General Hospital March 10, 1930, from a fracture of the skull received in an automobile collision in Cambridge the previous day.

The son of John C. McClure, he was born in Lawton, Oklahoma, 36 years ago, took an A.B. at Drury

College, Missouri, in 1916 and an A.M. at the University of Illinois in 1919. Graduating from Harvard Medical School in 1923 he served as surgical house officer and then as surgical resident at the Massachusetts General Hospital and engaged in private practice. He was a Fellow of the Massachusetts Medical Society.

## NEWS ITEM

BOSTON CONTRIBUTORS TO A MEETING OF THE NEW YORK ACADEMY OF MEDICINE—On March 18, the New York Academy of Medicine will devote an evening to diabetes as outlined in the following program:

The treatment of diabetes as influenced by—

a. New aspects in its pathology (15 minutes), Shields Warren, Boston (by invitation).

b. Diabetes in childhood (15 minutes), Priscilla White, Boston (by invitation).

c. The growing importance of pernicious anemia and coronary disease as complications (15 minutes), Howard F. Root, Boston (by invitation).

d. The end result of patients undergoing treatment between 1898 and 1929 (20 minutes), Elliott P. Joslin, Boston (by invitation).

Discussion: Frederick M. Allen, Nellis B. Foster, Henry R. Geyelin, Herman O. Mosenthal.

## NOTICE

NOTICE OF EXAMINATION FOR ENTRANCE INTO THE REGULAR CORPS OF THE UNITED STATES PUBLIC HEALTH SERVICE

Examination of candidates for commission as Assistant Surgeon in the Regular Corps of the U. S. Public Health Service will be held at the following-named places on the dates specified:

| | |
|---|---|
| At Washington, D. C., | May 5, 1930. |
| At Chicago, Ill., | May 5, 1930. |
| At New Orleans, La., | May 5, 1930. |
| At San Francisco, Cal., | May 5, 1930. |
| At Stapleton, S. I., N. Y., | May 5, 1930. |

Candidates must be twenty-three years and not over thirty-two years of age. They must have been graduated in medicine at a reputable medical college, and have had one year's hospital experience or two years' professional practice. They must satisfactorily pass oral, written, and clinical tests before a board of medical officers and undergo a thorough physical examination.

Successful candidates will be recommended for appointment by the President, with the advice and consent of the Senate.

Request for information or permission to take this examination should be addressed to the Surgeon General, U. S. Public Health Service, Washington, D. C.

H. S. CUMMING, *Surgeon General.*

## REPORTS AND NOTICES OF MEETINGS

### CLINICAL-PATHOLOGICAL CONFERENCE YALE MEDICAL SCHOOL

CASE I—History presented by Dr. Blake.

An exceedingly obese colored woman was admitted to "Isolation Wards" on January 18, 1930, complaining of cough with expectoration.

Her present illness dated back to December 12, 1929, at which time she had nausea, vomiting, fever and drenching sweating periods. These signs persisted for nine days, and forced her to remain in bed. On December 21, feeling better, she went back to work. However, the return of her previous symptoms besides a severe, almost constant cough necessitated her returning to bed. She remained in bed until her admission to the hospital on January 18. On admission her temperature was 102.2°, pulse 104, respirations 22, Blood Pressure 135/85, red blood cells 3,700,000, Hemoglobin 65% (Sahli), white blood cells 15,600. Urine and sputum were negative for Tubercle Bacilli. Throughout the whole period there was no history of upper respiratory infection and no history of hemoptyses or pleurisy. Her family history was negative for tuberculosis.

Physical examination revealed signs that were interpreted as bilateral pulmonary tuberculosis with coarse râles in the right upper lobe and consolidation of the right lower lobe. Abdominal examination revealed nothing. On the second day in the hospital the signs were interpreted as empyema of the right chest. Several thoracenteses revealed no fluid. On her first day in the hospital it was necessary to take x-ray pictures in the prone position. Those plates were unsatisfactory. On the second day x-rays were taken in the upright position. On this day the patient had a sudden attack of coughing and died before the final x-rays which demonstrated a subdiaphragmatic abscess, were developed.

NECROPSY FINDINGS (presented by Dr. Du Mortier with the aid of lantern slides): The abdominal cavity was opened and no free fluid was present. The appendix was in a retrocecal position. It was swollen and red. The tip of the appendix was gangrenous and showed an area of perforation surrounded by a thick purulent material which was followed beneath the ascending colon, quadratus lumborum, and parietal peritoneum to the postero-lateral surface of the liver. Between the right lobe of the liver and the inferior surface of the diaphragm was a large accumulation of foul smelling, sanguino-purulent fluid. About one liter of the fluid gushed out. The accumulation of fluid had pushed the diaphragm to the third interspace. The liver was attached to the inferior surface of the diaphragm by a fibrino-purulent exudate which was separated with ease. The right dome of the liver was thickened and edematous. The liver extended 5.5 cm. below the costal margin in the right mammillary line, 7 cm. below the xiphoid and 2.5 cm. below the left mammillary line. On removing the thoracic wall the left pleural cavity was found free from fluid and ad-

hesion. The right lung was bound down by adhesions covering the entire right base. The adhesions were of recent origin and were removed with ease. The diaphragmatic pleura was adherent to the superior surface of the diaphragm.

LUNGS — The right pleura contained a fibrinopurulent exudate throughout. The lower and middle lobes were dense and non-crepitant. The upper lobe was not unusual. On cut section the two lower lobes were solid and no alveoli could be made out. Around the bronchi were dark red areas. Microscopically the left lung showed dilated alveoli but no evidence of exudate or congestion. The right lung showed in the lower lobe a greatly thickened, edematous pleura, greatly dilated and congested blood vessels and a dense exudate composed of polymorphonuclear leukocytes, large and small mononuclear cells, and plasma cells. Large and small areas of hemorrhage were present. The exudate in the pleura was continuous with the exudate in the diaphragm. The alveoli were filled with a polymorphonuclear cell exudate. The alveolar walls were edematous and congested. The right upper lobe showed no pneumonic process but showed dilated alveoli.

THE LIVER—At the superior surface was a dense walled-off exudate which has undergone organization and has compressed to some extent the hepatic architecture which appears as a dense purple-brown tissue. On cut section the liver showed no evidence of abscesses. Microscopically the capsule of the liver was greatly thickened due to edema and a profuse exudate of polymorphonuclear cells, large and small mononuclear cells and plasma cells. Deeper in the hepatic architecture the necrosis was not so marked but there was some infiltration of polymorphonuclear and lymphocytes present. The diaphragm was thickened due to edema. The muscle fibers stained as dense homogeneous bands. The exudate was the same as in the lungs.

THE APPENDIX — Microscopically the lumen was found dilated and filled with polymorphonuclear cells. The mucosa was mostly necrotic. The mucosal stroma and the submucosa were filled with a polymorphonuclear cell exudate.

### DISCUSSION

DR. HUSSEY: Anatomically the process is evident. The infection extended upward from the perforated appendix. This was a cellulitis, not a localized peritonitis. Subdiaphragmatic abscess may occur on the right or left side. There is more difficulty with the diagnosis and pathogenesis when on the right side. In the last two thousand cases which have come to autopsy at the New Haven Hospital, six had subdiaphragmatic abscess. Two of these cases were in children, under two years of age. In adults all except this case were diagnosed.

DR. LA FIELD: The first x-ray film was taken in the prone position and was unsatisfactory. There was an absence of all markings of the right lower lobe and a complete consolidation below the second interspace. There was no shifting of heart to the left side. The left diaphragm was normal. The second film, taken on the day of death, was in the upright position. There was an apparent fluid level in the right lung field. There was an "air bubble" between the diaphragm and liver on the right side.

DR. OUGHTERSON: From a surgical point of view the diagnosis of subdiaphragmatic abscess gives much concern. X-rays may be taken at different angles to see if nodules are in liver or diaphragm. Gas may or may not be produced by the organisms. Lateral x-ray films aid in differentiating cysts, anterior or posterior position of the abscess, and the general extent of the involvement. Patients should not be kept in a supine position. They should be kept upright. With a subdiaphragmatic abscess the exploring needle may invade the pleural cavity. Withdrawal of the needle may cause extension of the process from below the diaphragm to the pleural cavity and cause an empyema. If pus is obtained the operation should be done, immediately, before the needle is taken out. One-half the subdiaphragmatic abscesses are the result of an extension of the process from the stomach or appendix. Without treatment ninety per cent. die. With drainage the mortality is 30%.

DR. BLAKE: The diagnosis of appendicitis was not considered because of incorrect emphasis of some features of the history. During the first three weeks there were abdominal symptoms—nausea and vomiting but no abdominal pain. During the last three weeks the symptoms were respiratory. There was a leukocytosis of 15,600 but no increase in the polymorphonuclear leukocytes. Such a picture may be found in tuberculosis but with an abscess of many weeks' duration there may be a leukocytosis with no increase in polymorphonuclear leukocytes. If this patient had been put in an upright position she would have been better off.

### ANATOMICAL DIAGNOSIS

Primary: Acute Appendicitis (retrocecal), with perforation; subdiaphragmatic abscess, diaphragmitis; fibrinous pleurisy; atelectasis and focal pneumonia (right middle and lower lobes).

Subsidiary: Generalized arteriosclerosis; fibromyomata of the uterus; obesity.

PATIENT II.—History presented by Dr. Treiheit.

The patient was a thirty-four-year-old white housewife admitted to the gynecology service with a complaint of pain in the left hip and lower back. She was referred to the hospital by a physician in Stamford with a diagnosis of "malignancy".

The patient was well until two and one-half months before admission to the hospital. At that time she began having pain in the region of the left hip and left groin. Examination by her physician revealed tenderness over the left hip and limitatfon of motion of the left hip joint. The patient was given treatment for "arthritis" by her physician. No improvement having followed, she had x-rays taken two weeks later which disclosed rarefaction of the left pubic bone. Further examination revealed an enlarged and indurated clitoris with a nodular mass and an "elastic swelling" in the left pubic bone. Her physician believed that she had a malignant tumor

somewhere which metastasized to the pelvic bones. There was no history of menorrhagia or metrorrhagia. No pain or difficulty on urination. Menstruation was normal up to the time of admission.

Physical examination revealed an emaciated, nervous, restless patient, moaning and complaining of considerable pain in her back. In the abdomen there was a firm, irregular mass which extended about four c.m. above the symphysis. Rectal examination revealed a firm cord-like structure in the left side of the rectovaginal septum and extending laterally. The mucosa of the rectum was normal. In the left lateral vaginal wall several hard nodules were present. Examination of the extremities revealed great pain in the left inguinal region on stretching the adductor muscles.

Course in Hospital—An operation one week after admission for examination and curettage revealed nodular masses in the clitoris, areas of induration around the vaginal orifice, enlarged body of the uterus and nodular masses in the region of the cul-de-sac. A sound passed into the uterine cavity suggested an enlargement of the cavity. The uterine cavity was curetted. Biopsy from the uterine cavity showed a picture of adenocarcinoma. Sections from the vaginal nodules also showed adenocarcinoma. The patient made a good postoperative recovery. She complained of a severer pain in the pelvic region and it became increasingly difficult to relieve her of the pain in spite of large doses of morphia, chloral hydrate and magnesium sulphate. Four x-ray treatments gave only slight relief. Red blood cell count was 3,500,000. Hemoglobin 88 per cent. Tallqvist. Four weeks after admission her pulse was 110 and respirations 25 per minute. Five weeks after admission the condition of the patient was much worse. Pain was extremely severe and her color was an ashen gray. Five and one-half weeks after admission the patient became comatose and irrational. Temperature rose to 105°, pulse 150, respiration was slow and shallow and the patient died. X-rays of pelvic bones disclosed destruction of the superior and inferior rami of the left pubic bone with no evidence of bone regeneration. X-rays of chest, vertebrae, bladder, uterus and kidneys were normal.

Necropsy Findings (presented by Dr. Finley with the aid of lantern slides): On exposing the abdominal cavity the most striking change was the great size of the liver. Beneath the liver capsule and peritoneal lining are large numbers of hard white single and confluent nodular masses. The greater omentum is bound down to the lower portion of the anterior abdominal wall and to the tumor projecting above the symphysis pubis. Metastatic nodules are present in the lower portion of the abdominal cavity, scattered beneath the peritoneal lining. These masses are most prevalent around the pelvic brim. Examination of the large mass palpated before opening the abdominal wall discloses a hard solid mass with a nodular surface and an occasional cyst containing a dark red-bluish colored fluid. This mass as it lies in the mid line appears to be continuous with the fundus of the uterus. However, on lifting this mass a normal uterus is found lying beneath. The tumor mass extends to the right and is continuous with what apparently was at one time the right ovary. Beneath both parietal and visceral pleurae are found many small white glistening nodular masses. While removing the thoracic organs, all the hilic lymph nodes are found to be involved by metastatic tumor growths.

Liver—About one-half the weight of this organ is due to metastatic growths. The liver weighs 3,600 grams. Scattered over its entire surface but most prominent on the anterior surface are large numbers of confluent and single gray-white translucent nodular metastatic tumor masses. Microscopic sections through the metastatic areas showed a characteristic squamous cell type of carcinoma. Small masses of these tumor cells are walled off by connective tissue. Many of the squamous cells form long papillae reaching into the stroma of the growth. In the large metastatic nodules the amount of keratinized cells is more abundant than in the smaller nodules. Fine strands of connective tissue can be seen winding their way in between the keratinized areas, suggesting that the connective tissue stroma is replacing these more adult tumor cells.

Pelvic Organs—Besides the right ovary the left ovary was involved in the malignancy. The uterus was carefully examined for evidence of cancer but none was found. Microscopically the tumor of the right ovary consists mostly of connective tissue stroma in which lies scattered nodular masses of squamous cells, most of which have undergone keratinization and have a hyalinized appearance. The absence of large well stained squamous cells in the abundance of connective tissue stroma and keratinized epithelium is quite striking. The wall of the cyst essentially consists of connective tissue within the outer layer of which lies keratinized squamous celled epithelium.

Lungs—Palpation and cross sections of both lungs disclosed small nodular masses lying immediately beneath the pleurae and in the parenchyma of these organs. Masses are more numerous in the bases of both lower lobes. Microscopic sections through the nodules showed a squamous type of carcinoma.

Spleen—At the upper pole, lying immediately beneath the capsule is a pale white-yellowish translucent mass. On cross section and microscopically it resembled the other metastatic nodules.

Kidneys—Immediately beneath the capsule of the left kidney is a metastatic nodule. Microscopically it shows a squamous type of tumor cells.

Lymphatic Glands—Besides the hilic glands the inguinal, retroperitoneal nodes, and especially those along the junction of the hypogastric artery were greatly enlarged by metastatic nodules which on microscopic section showed a squamous type of cancer cell.

Bones—A mass extends from the inner aspect of the obturator foramen, eroding away the pubic and lower rami of the ischium. The body of the second and fourth lumbar vertebrae are involved by metastases.

### Discussion

Dr. Hussey: This is a rare type of tumor—a primary squamous cell epithelioma of the ovary. The germinal epithelium became metaplastic. This tumor

is extremely malignant in its metastasis—as are also hypernephroma and carcinoma of the testicle. This type of tumor is one of the class which has a tendency to metastasize to bone.

CLINICAL DIAGNOSIS

Carcinoma of the body of the uterus with extensive metastasis.

ANATOMICAL DIAGNOSIS

Primary: Adenocarcinoma of ovary (bilateral); with metastasis to regional lymph nodes, obturator foramen (left); peritoneum, liver, spleen; kidney (right), pleurae, lungs, hilic lymph nodes, vertebrae (2L, 4L).

Subsidiary: Calculi of kidney (right); cystitis.

## THE ACADEMIC CAREER IN SURGERY

Under the auspices of the Phillips Brooks Association on Monday, February 24, 1930, Dr. Elliott Cutler, Professor of Surgery at the Western Reserve Medical School and Lakeside Hospital, Cleveland, Ohio, spoke on "The Academic Career in Surgery."

Dr. Cutler first pointed out that the academic surgeon is not doing better work than the general practitioner, only a different kind of work. Furthermore, it is not a sheltered life. His responsibilities extend to all the patients on his service whether he gives them his personal attention or not. He is called on by his colleagues for advice even in the simplest matters. His trustees disturb him at all hours. There are his students, both pre- and post-graduate, whom he must train by precept and by personal contact. Then, after he has attended to such hospital affairs of organization as are necessary, he must spend the remaining few moments in reading to keep abreast of the swiftly moving streams of medical progress.

The rewards of such a life are rich, even though it is largely a matter of whether one can stand the pace. The chief compensation, said Dr. Cutler, may be counted in the success and development of one's pupils. He may find great satisfaction in accomplishing some individual work of experimental type himself. Finally, he will have that happiness of being able to care for and to assist a great number of stricken individuals.

The methods which prepare one for such an academic life in Surgery and its rewards are various. No one course, alone, will always satisfy. However, there are certain necessary things. First, you must become expert in the art of surgery, and this only through practice. The minimum period of training is five years. The candidate must also master the technique of teaching. He must have experience in laboratory and investigative methods. Other qualifications such as diplomacy and ability to inspire confidence, and to work out important problems in the advancement of medical science must perforce vary with his personal qualifications.

Finally, Dr. Cutler made clear that whereas formerly there was a clash between the laboratory man and clinician, now both are working in harmony. The result is an ideal atmosphere in which the medical student can acquire the necessary education.

## THE CUTTER LECTURE

The Cutter Lecture on Preventive Medicine was delivered by Lafayette B. Mendel, Ph.D., Sc.D., Sterling Professor of Physiological Chemistry at Yale University, on Friday, February 28, 1930 in Amphitheater E of the Harvard Medical School. His subject was "Nutrition and Growth."

The recent enthusiasm, said Professor Mendel, in hygienic and prophylactic living is fundamentally based on nutrition and growth. Growth, upon which the maintenance of the race depends, is a correlation of form and composition. It is defined as a multiplication, enlargement and deposition of cells. It is not uniform throughout life, the per cent. of growth declining from birth onwards. The factors which affect it are environment, food, and inherent tendencies.

The question of underweight is still a problem. Recent studies have shown that students today are larger and weigh more than they did twenty years ago. The body does not grow unless all the essentials are present. Neither does it grow normally unless all parts can grow together, thus preventing deformities. The law of the minimum is then postulated which says that the growth of the body does not grow more rapidly than the supply of any single essential permits. Professor Mendel gave some measure of consolation to persons of small build by stating that rats which have been stunted in their body growth relearn a "maze problem" faster than their normally growing litter mates!

A careful proportion in the diet is not now considered so essential because the body can adapt itself readily to excesses. Neither fat nor carbohydrate lack will stunt growth as long as sufficient calories are present and no essentials are lacking. Thus, as far as growth is concerned, any proportion will do if the essentials are present, though other dyscrasias may supervene. There are several essentials. Nitrogen cannot be synthesized by the body and thus a sufficient intake is necessary. Of the amino acids only four,—cystine, lysine, tryptophane, and histidine,—are now considered to be essential. These cannot replace each other, and though they form only a few parts per million of the body, their lack spells nutritional disaster. Vitamins in the diet occupy an essential position.

There are, however, periods of growth cessation without discoverable dietary deficiencies. Whether these are physiological is unknown. The capacity of growth is not lost by prolonged failure to grow. Yet, abnormalities in growth may be due to constitutional or hereditary diatheses. Here diet will make little difference. The cause may be endocrine, and here the speaker described the clinical entities of cretinism and gigantism.

Professor Mendel concluded that the science of nutrition is of immense importance, and while much of its mystery may be impenetrable, gross ignorance of it is inexcusable.

## THE GREATER BOSTON MEDICAL SOCIETY

A joint meeting of the Greater Boston Medical Society and the Beth Israel Hospital was held on Tuesday, March 4, 1930 at the Beth Israel Hospital. Dr. Harry Solomon presided and introduced the speakers.

The first paper on "The Present Status of the Hinton Test" was read by Dr. Arthur Berk. He described the basic features of the Hinton test and spoke of a modification not yet published which is 25% more sensitive than the original. He told of a study now in progress in which 2000 cases are being analyzed in the endeavor to find some sure way to state whether a cure has been attained without doing a lumbar puncture. He expects to prove that the modified Hinton test is 100% reliable in the diagnosis of neurosyphilis.

Dr. J. Kasanin spoke on "A Study of Social Factors in Mental Diseases." He pointed out that present day theories postulate either an organic or functional basis for mental diseases. He and his collaborators are making a study to determine whether the "shut-in" type always develops schizophrenia, and the emotionally unstable individual develops a manic-depressive psychosis. He outlined their method and described a questionnaire which is being used in the research.

The third paper on "The Use of Stramonium in Parkinsonism" was presented by Dr. A. H. Braverman. Many drugs, including intravenous sodium salicylate, gold, silver, atropine and pilocarpine, have been tried. He reported results in the treatment of eleven cases of post-encephalitic parkinsonism with stramonium. The patients showed a less mask-like face, and the range of voluntary movement increased, though the tremor was not affected. The dose was twenty minims three times a day, increased daily five minims until the patient was getting seventy minims four times a day. Toxic symptoms of blurred vision and dry mouth occurred at thirty minims three times a day or more. The nausea disappeared as the dose increased. Hyoscine relieved the tremor. He concluded by pointing out that stramonium is only palliative, and not a cure.

Dr. William Dameshek told about the "Blood Findings in General Paresis." There is a monocytosis of 10% or better in 50% of the cases. Histocytes are seen in two per cent. of cases. Lymphocytosis with a predominance of large lymphocytes is a constant finding. Plasma cells are seen in 27% of cases. Toxic changes such as vacuolization, fewer granules, and irregular staining are found in 80% of cases. He found the most marked changes in degenerated paretics in bed and almost normal blood in patients only slightly euphoric. He concluded that the work has no practical value as yet.

The fifth paper on "Bio-chemistry of Brain Activity by Internal Jugular Puncture Method" was presented by Dr. Abraham Myerson. Reference has been made to this subject in a previous issue (*N. E. J. M.*, Feb. 27, 1930, page 458).

The last paper on "The Effect of Inhalation of Carbon Dioxide in Certain Neuropsychiatric States" was read by Dr. Harry C. Solomon. Motion pictures were shown of patients before and after the administration of carbon dioxide. The method has been used in benign stupors, catatonic states, and in the Parkinson syndrome. The convulsions and tremor increased during the inhalation, but completely disappeared after three minutes of breathing the carbon dioxide mixture. Dr. Solomon stated that not all that happens is known. What we do know is that there is a dilatation of cerebral vessels, a faster cranial circulation, and an increased intracranial pressure up to 600 mm. of water. The method needs further study.

The meeting was well attended and followed by refreshments.

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## HARVARD MEDICAL SOCIETY

The Harvard Medical Society held a meeting on Tuesday, Feb. 25, 1930 at eight o'clock in the Peter Bent Brigham Hospital Amphitheatre. The following described cases were presented:

The first case was presented by Dr. Glenn of the surgical service. The patient was a 72-year-old stone-cutter who was admitted following a rupture of an abscess in his inguinal region. A brother died of "stone-cutter's consumption" twelve years before and the patient himself had to stop work five years before because of "intestinal flue." One year ago he had a pulmonary hemorrhage with no sequelae. Six weeks before admission he developed pain in the lower abdomen without apparent cause. It soon localized in the right lower quadrant and he noticed a mass which increased in size, but was not unduly painful. The morning of admission the mass suddenly ruptured and discharged a cupful of pus. Physical examination on entrance showed a man with evidences of loss of weight. The right chest was depressed, with dulness, increased fremitus and breath sounds throughout. The heart was pulled to the right. In the right inguinal region was fullness, redness, induration, and a sinus which discharged pus on pressure. He showed a red count of three million, a white count of ten thousand with a lymphocytosis of 30%, and many acid-fast bacilli in the sputum. X-rays of the chest showed fibrosis at both apices with cavitation at the right apex, and the heart pulled to the right. Pictures of the spine were negative. Lipiodol injections of the sinus tract showed the cavity to spread laterally and to end in blind pockets. Barium enema was normal. The patient was presented for diagnosis.

The medical case was presented by Dr. Tollman. The patient was in the hospital for the thirty-second time, with the same complaint as before, that of cough and shortness of breath on exertion. His trouble began five years before his first admission when he developed a cough which grew steadily worse. After a cold of three weeks' duration he developed dyspnea on exertion. In the hospital dulness was found at the apices, râles throughout the lungs, and an irregular heart. Digitalis therapy helped his general condition greatly. The cough persisted and his sputum showed many acid-fast bacilli. He was sent to a sanatorium which he left against advice. In 1922 he was noticed to have

dilated blood vessels in his back originating in the axillae and extended obliquely towards the sacrum. X-ray of the heart showed a dilated vigorously pulsating aorta, and indentations of the ribs which had been eroded by enlarged vessels. The diagnosis was coarctation of the aorta.

After the presentation of cases Dr. H. A. Christian introduced Dr. William L. Moss, Assistant Professor of Bacteriology and Immunology at the Harvard Medical School. He spoke on some aspects of the cruise of the Illyria and illustrated his travelogue with many lantern slides.

The purpose of the expedition, said Dr. Moss, was to collect specimens for the Field Museum of Natural History of Chicago. The trip lasted about a year.

Beginning in Boston in September, 1928, the expedition sailed to Bermuda and Cuba, then through the Panama Canal, and on to the South Sea Islands from Galapagos to Java. Several interesting incidents were reported. The island of New Hebrides is under joint control of the British and French, the resulting situation being very unsatisfactory. The wealth in this island is measured in pigs, the wives ranging in value from 1-30 pigs. It is easier to teach the natives so-called "pigeon English" than to try and learn the native dialect. A peculiar belief of the natives is that if they so will they can lie down and die in 48 hours. Dr. Moss spoke of many other interesting happenings the charm of which is lost in transcribing them to paper.

The meeting was very well attended and a pleasant change from the usual scientific discussions.

## JOINT MEETING OF THE GREATER BOSTON MEDICAL SOCIETY AND THE BETH ISRAEL HOSPITAL

A joint meeting of the Greater Boston Medical Society and the Beth Israel Hospital was held on Tuesday, February 4, 1930, at 8:15 P. M. at the Beth Israel Hospital. Dr. Solomon introduced Dr. Albert A. Epstein who spoke on "A Consideration of the Present Day Problems in Diabetes Mellitus."

Dr. Epstein pointed out that today we have turned from the clinic to the laboratory in our researches. Diabetes has become a study of grams and calories, instead of the patient. It is better to consider the patient and his reactions rather than his intake and output.

Diabetes is looked upon as a perversion of carbohydrate metabolism, a deficiency in its storage, and combustion which leads to a non-utilization of fats. The acidosis which results may cause an impairment of the kidneys as well as further derangement of the carbohydrate utilization. If the kidneys are diseased, the amount of sugar in the urine is no indication of the patient's condition, and Dr. Epstein stressed the value of blood sugar determinations. Persistent hyperglycemia without a tendency to rise should be untreated because insulin in these cases brings unfavorable responses. There are also some cases with a high blood sugar in which symptoms of insulin shock may develop. The relation of the blood sugar to vascular damage is still a question.

Coma is of two kinds. The ketogenic type caused by an imperfect combustion of fats should be controlled by administration of carbohydrate and insulin. The non-ketogenic type should be treated with alkalies intravenously.

Diabetes is no contra-indication to surgery, especially surgery with infection present. To put off operation to treat the diabetes is useless because it will have no effect on the post-operative course. The presence of infection makes the diabetic treatment more difficult because infection reduces the acidosis and increases the utilization of insulin so that more has to be given. Infections in diabetes spread more easily, but they are less virulent than in non-diabetics, due, Dr. Epstein thinks, to the fact that bacteria lose their virulence on sugar containing media. Infectious foci should be searched for. In any case of diabetes not doing well, one must suspect hyperthyroidism which in these cases shows no signs except a high metabolic rate. In the presence of infection, fats should be eliminated. This gives a low caloric diet; the condition is usually temporary.

The presence of sugar in the urine does not necessarily mean diabetes. Dr. Epstein showed that levulose, often seen in children, and pentose, seen in young adults, are of no diabetic significance.

The story of diabetes is not complete. We have often found no cause or cure for diabetes, only a method of its control. The degeneration of the Islands of Langerhans may be the result and not the cause of diabetes. People in coma do not die of lack of insulin because at autopsy the organs are full of insulin. Thus, Dr. Epstein queried, "Is the disease due to a decrease in the production or the presence of inactive insulin?" The theory he proposed to explain this is as follows. There is a primary change in the permeability of the capillary walls, from the result of which the trypsin combines with the insulin in the blood stream making it inactive. The resulting product is carried to the liver where glycogenolysis results and a high blood sugar occurs.

The paper was followed by slides showing some interesting cases of Dr. Epstein. The discussion was opened by Dr. Baker, and ably carried on by Dr. Rudy and Dr. E. P. Joslin.

## NEW ENGLAND PHYSICAL THERAPY SOCIETY

SECOND REGULAR MEETING

Date: Wednesday, March 19, 1930.

Place: Boston Square and Compass Club, 488 Beacon Street, Boston, Mass.

Time: Dinner at 6.30 P. M. Scientific session at 8 P. M.

Paper: By Arthur L. Brown, M.D.

Subject: The Place of Electro-Therapeutics in General Practice.

The dinner is to be a special feature of the meeting. There are to be some good speakers. The progress of the Convention will be learned from the Committee Chairmen. A travelogue film will show some of the scenes en route to the next Physiotherapy Congress at Liége, Belgium. President Wilcox is

anxious to have a good attendance. The council will meet at 5.30 o'clock just preceding the dinner to pass upon applications and to transact any other business. Councilors please attend.

ARTHUR H. RING, *Secretary.*

### NEW ENGLAND ROENTGEN RAY SOCIETY

The March meeting of the New England Roentgen Ray Society will be held at the Boston Medical Library, March 21, 1930 at 8:15 P. M. The Business meeting will be at 8:15 P. M. followed by a Scientific Session. Twenty minutes will be allotted for members to demonstrate interesting cases. Dr. Frederick T. Lord of Boston will give a paper on "The Relative Value of Clinical and X-ray Findings in Certain Bronchopulmonary Conditions." The usual dinner will be held at the Harvard Club.

T. R. HEALY, *Secretary.*

### BOSTON MEDICAL HISTORY CLUB

Boston Medical Library, 8 The Fenway.

Monday evening, March 17, at 8.15 o'clock.

Experiences of a Medical Book-Hunter in Boston—Dr. A. B. Lyon. Discussion by Rival Book-Hunters.

Sir John Harington—Dr. I. H. Coriat.

Alfred Nobel: Medical Incidents and Nitroglycerin—Dr. W. P. Coues.

JAMES F. BALLARD, *Secretary.*

### CLINIC AT THE BOSTON CITY HOSPITAL

A clinic will be held at the Boston City Hospital, Wednesday evening, March 26, 1930 at 8:15 P. M. in the Cheever Amphitheater.

PROGRAM

The Injection Treatment of Varicose Veins, Dr. H. O. McPheeters, Minneapolis, Minn. Discussion by Dr. E. E. O'Neil, Dr. A. W. Allen, Dr. John Homans.

Doctors and medical students are cordially invited.

COMMITTEE ON HOSPITAL CLINICS.

### NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

The monthly Clinical Conference of the New England Hospital for Women and Children will be held at the hospital, Dimock Street, Roxbury, on Thursday, March 20, at 8 P. M. The following cases will be presented by members of the staff: Carcinoma of the uterus with broncho pneumonia and diabetic coma; Compound comminuted fracture of the skull; Intracranial hemorrhage of the new-born; Extreme malnutrition.

ALICE H. BIGELOW, M.D., *Secretary.*

### PLYMOUTH DISTRICT MEDICAL SOCIETY

The regular meeting of the Plymouth Medical Society will be held at the Moore Hospital, 167 Newbury Street, Brockton, Mass., Thursday, March 20, 1930, at 11 A. M.

Election of a Nominating Committee for the ensuing year.

Program: Acute Intestinal Obstruction.

1. X-Ray Studies of Acute Intestinal Obstruction and Lesions of the Small Intestines. Dr. Max Ritvo, Consulting Roentgenologist, Moore Hospital.

2. Surgical Treatment. Dr. G. A. Moore.

3. Diagnosis; Preoperative and Postoperative Treatment. Dr. Chester Jones, Physician to Massachusetts General Hospital.

Luncheon will be served at 1 P. M.

## SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

**1929-1930**—Massachusetts Dietetic Association. Complete schedule appears on page 852, issue of October 24.

**March 13**—Massachusetts General Hospital. Complete notice appears on page 510, issue of March 6.

**March 13**—Lecture by Professor Hecht at the Harvard Medical School. Detailed notice appears on page 459, issue of February 27.

**March 17**—Boston Medical History Club. Complete notice appears elsewhere on this page.

**March 19**—New England Physical Therapy Society. Detailed notice appears on page 555.

**March 20**—New England Hospital for Women and Children. Complete notice appears elsewhere on this page.

**March 21**—New England Roentgen Ray Society. Complete notice appears elsewhere on this page.

**March 26**—Clinic at the Boston City Hospital. Complete notice appears elsewhere on this page.

**March-October** — International Medical Postgraduate Courses in Berlin. Complete notice appears on page 853, issue of October 24.

**April 14-18**—The New England Public Health Institute. Complete notice appears on page 55, issue of January 2.

**May 2—Annual Meeting of the American Society of Stomatologists. Detailed notice appears on page 92, issue of January 9.**

**May 5-10—First International Congress on Mental Hygiene. Detailed notice appears on page 93, issue of January 9.**

**September 4-8—Fifth International Congress on Physiotherapy. Complete notice appears on page 906, issue of October 31.**

**October 27—The American Public Health Association. Detailed notice appears on page 1271, issue of December 19.**

### DISTRICT MEDICAL SOCIETIES

#### Essex North District Medical Society

**May 1**—Thursday—Censors' meeting at Hotel Bartlett, 95 Main Street, Haverhill, (Telephone 3430) at 2 P. M. sharp. Candidates should present their diplomas to the Secretary one week in advance.

**May 14**—Wednesday—Annual Meeting, at Anna Jaques Hospital, Highland Avenue, Newburyport, Mass., at 12 noon. Subject, "Infective Foci: Sense or Nonsence." There will be talks and discussions on the subject from individual points of view, by seven physicians engaged in nine of the specialties of medicine, and from the dental standpoint.

J. FORREST BURNHAM, M.D., Secretary.

#### Essex South District Medical Society

**Wednesday, April 2, 1930**—Hotel Hawthorne, Salem. Dinner at 7 P M. Speaker: Dr. Timothy Leary, Boston. Subject: "Diagnostic and Therapeutic Pitfalls in Intracranial Pyogenic Infections."

**Thursday, May 1, 1930**—Censors meet for Examination of Candidates at Salem Hospital at 3:30 P. M.

**Tuesday, May 13, 1930**—Annual Meeting. The Tavern, Gloucester. Speaker: Dr. C. Macfie Campbell, Director of Boston Psychopathic Hospital. Subject to be announced later. Ladies invited. Dancing.

R. E. STONE, M.D., Secretary.

#### Franklin District Medical Society

The next meeting of the Franklin District Medical Society will be held at the Weldon Hotel, Greenfield, Mass., on the second Tuesday of May, at 11 A. M.

CHARLES MOLINE, M.D., Secretary.

#### Hampshire District Medical Society

**April 9**—This meeting will occur at 11 A. M. and will be held at the Dickinson Hospital, Northampton.

The Annual Meeting will be held in May.

LUTHER O. WHITMAN, M.D., Secretary.

**Middlesex East District Medical Society**

**May 13**—At Unicorn Country Club, Stoneham.

ALLAN R. CUNNINGHAM, M.D., Secretary.

**Middlesex South District Medical Society**

**April 16, 1930**—Annual Meeting. Commander Hotel, Cambridge. Program to be announced.

**May, 1930**—Censors' meeting.

ALEXANDER A. LEVI, M.D., Secretary.

**Norfolk District Medical Society**

**March 24, 1930**—Roxbury Masonic Temple. Dr. Burton E. Hamilton. Subject to be announced.

**May 1, 1930**—The Censors meet for the examination of candidates, in the Roxbury Masonic Temple, 171 Warren Street, Roxbury, at 4:00 P. M.

Applications must be in the hands of the Secretary at least one week previous to date of examination.

**May 6, 1930**—Annual Meeting. Program to be announced.

Owing to inability to engage the Roxbury Masonic Apartments on the last Tuesday of the month, such meetings as will be held in the Roxbury Temple will be held Monday evenings as per the dates above.

FRANK S. CRUICKSHANK, M.D., Secretary.

**Plymouth District Medical Society**

**March 20, 1930**—Moore Hospital, Brockton. Detailed notice appears on page 556.

**April 17, 1930**—Annual Meeting, Commercial Club, Brockton, 7 P. M. Election of officers. Annual oration, Dr. Frederick F. Weiner, "The Management of Prostatic Diseases," with moving pictures.

G. A. MOORE, M.D., Secretary.

## BOOK REVIEWS

*The Physician Throughout the Ages.* By ARTHUR SELWYN-BROWN, B.S.C., M.A., Ph.D., LL.D. New York: Capehart-Brown Company, Inc., 1928.

This elaborate work, in nine books and two large volumes,—a total of over seventeen hundred pages,—embodies a record of medicine from the earliest historical period, embracing a general survey of medical practise, the social history of the doctor, with medical chronology and biographies of outstanding physicians. Its purpose is to follow the development of the physician from the remotest times to the present, with sufficient detail to enable the reader to form his own conclusions regarding the status of the profession at any given period. Like Wells's *Outlines of History* the book begins with a study of the formation of the earth, the evolution of man, and the early evidence of paleopathology. The editor is apparently author of much of the first volume, to which, however, there are contributions by forty distinguished collaborators, while there are seventy-five contributors to volume two. Much space is devoted to brief biographies of noted practitioners since classical times and there are many sketches of prominent American physicians of the past century. Altogether there are over one hundred and sixty portraits of distinguished physicians and a hundred and forty-four general illustrations of extreme and varied interest. Though representing the work of many hands, the work has an easy narrative style and its discursive, loosely knit character makes it agreeable and pleasant to read. What one thinks of most, however, in reviewing these lives and achievements of the leaders of medicine throughout the ages is the devotion, sufferings, and victories of the other unrecorded thousands whose labors have helped mankind and whose service was no less noble and self-sacrificing than that of the more conspicuous few whose names are remembered.

*An Outline of Endocrinology.* By W. M. CROFTON, B.A., M.D. Second Edition. William Wood and Co. Price $3.00.

This little book is written particularly for medical students and general practitioners, according to the preface. It should, therefore, contain only proven facts, leaving theory to the more elaborate systems of endocrinology.

Unfortunately, the author has not confined himself to this straight and narrow path. The sections on the histology, development, physiology and pathology are for the most part concise statements of the generally accepted ideas, and are open to criticism only on the ground that some of the newer work has not been included. The work of Allen, Doisy and Frank on the follicular hormone is a case in point.

The student or general practitioner, however, is more concerned with clinical entities than with the results of animal experiments, and the author's failure to include sections on the differential diagnosis of various endocrinopathies is the most serious defect in the book. It is generally recognized by those seriously interested in clinical endocrinology that the diagnosis of disturbances of glandular function, although they may be strongly suspected from the patient's story and physical condition, can be proven only by tests of vital function which disclose the present functional level of endocrine activity. Yet these tests receive scant mention throughout the work. And so valuable an adjunct to diagnosis as the basal metabolism determination is not mentioned in the chapter on the thyroid gland. The book conveys the impression that exact diagnosis is unimportant—an impression all too prevalent among those dispensing endocrine therapy.

The sections on therapy, however, do much to explain the above omission, since the treatment recommended is almost entirely symptomatic. For example, whole gland pituitary substance given by mouth is recommended as one of the best tonics known to the author "in neurasthenia, convalescence, exhaustion from overwork and sexual excess," in the latter case combined with testicular extract. Prima donnas, "in whom it is important to preserve the quality of the voice," should try ovarian extract. (If by any chance this treatment should fail, the Victor Record will preserve the voice.) The diabetic, growing weary of insulin injections, may turn to any one of "several products which have been on the market for many years which, given by mouth will enable the majority of patients to metabolise sufficient glucose to keep them with a feeling of well being. . . . It does not matter very much how much sugar the patient is excreting so long as he is metabolising enough to give him sufficient energy."

One grows weary trying to understand why fair white paper should be so maltreated. Books like this serve only to retard exact knowledge of a most important field in medicine, in which the truth, as it is occasionally glimpsed through the clouds of

imaginative literature, is fully as strange, and far more important than fiction.

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*Rickets Including Osteomalacia and Tetany.* By ALFRED F. HESS, M.D. Lea and Febiger, Philadelphia, 1929. Price $5.50.

This book presents the subject of Rickets and allied conditions in the light of what the author describes as our "newer knowledge" of this subject. Our conceptions of Rickets as a disease remained essentially unchanged until the last decade when, as the author states, new influences, namely, the discovery of vitamines and the importance of ultra-violet light brought a new interpretation of the cause and treatment of this disease. This book, therefore, is a very necessary and natural development of a medical subject which demanded a new presentation in the light of both previous observations and more recent discoveries. The author has developed the subject in a most exhaustive, painstaking and critical manner always leaving a definite conception of each phase of the subject. The first chapters deal with the history and geographic distribution of Rickets and there follow chapters on the Experimental Production, Etiology, Pathology and Pathogenesis. The chapter on Symptomatology of Rickets gives in a relatively few pages a classical description of the disease as understood to-day. The book gives authoritative information in regard to x-ray diagnosis, the use of Ultra Violet Light, Cod Liver Oil and Viosterol. The chapters on Osteomalacia and Infantile Tetany also bring these subjects up-to-date in a very careful, concise presentation. Of great value is the biliography containing all the important articles and arranged in chapters corresponding to the text.

This book is an important contribution to medical literature and will be of great interest to all students of nutritional problems in infancy.

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*The Blood Picture and Its Clinical Significance.* By PROFESSOR DR. VICTOR SCHILLING. Translated and Edited by R. B. H. Gradwohl, M.D. Seventh and Eighth Revised Edition. St. Louis. The C. V. Mosby Company, 1929. Price $10.00.

This book contributed by one of our great hematologists is now available in English. It is divided into four sections: (1) Technique; (2) Theory, morphology and division of the blood picture; (3) Fundamental principles for clinical use of the blood picture; (4) Selected examples on practical use of hemograms.

The real contribution of Schilling consists of the hemogram by which is meant the diagrammatic analysis of the blood picture. Special attention is directed to the neutrophiles which are divided into the myeloblasts, juvenile forms, "stab" cells and segmented forms, this being the order of their maturity. The appearance of the more immature forms is spoken of as "a shift to the left" or regenerative shift. "So far as the numerical proportions are concerned, the course of a regenerative shift is about as follows: the neutrophiles increase absolutely (total count increase); the increase is dependent upon the excess of 'juvenile' forms (regenerative shift); both processes increase to very high counts with a very high shift up to the formation of myelocytes. In the severest cases, the cell consumption begins to exceed the constantly increased regeneration (diminution of total count), together with further advancing shift—Anisohypoleucocytosis. The course of a degenerative shift is simpler: the newly introduced neutrophiles are poorly developed and partially not segmented. They are neither diminished nor increased. The degenerative influence leads to a central, myelogenous inhibition of growth and to a reduced importation, as well as to a relatively increased disintegration; thus the peripheral amounts are lowered and the shift proceeds under continuing degeneration. Most of the infectious shifts are mixed forms (mixed regenerative-degenerative picture)." Associated with this reaction there is commonly a disappearance or reduction of eosinophiles and a decrease in the number of lymphocytes; the reappearance of the eosinophiles and increase in the number of lymphocytes is indicative of improvement or recovery.

Aside from this important contribution, the book is disappointing. It could easily have been condensed into a monograph instead of a book of almost 400 pages. With regard to the formation of platelets Schilling states that "it is plausible and in accord with all observations to date to assume that they are derived from the nuclei of the nucleated primary erythrocytes." In this country the hypothesis regarding the formation of platelets most accepted is that of Wright which considers them as derived from the megakoryocytes. Furthermore, the important work of Howell and Cekada concerning the stability of platelets in hemophilia is not mentioned. In fact, the book gives one the impression that there has been very little hematological research outside of the author's country; this is further indicated by the choice of references. It is natural therefore for him to accept the Morawitz theory of blood coagulation rather than Howell's. In the treatment of thrombopenic purpura the author considers splenectomy as the last resort whereas it is generally advocated here early in the disease. In view of the existence of direct methods of red cell diameter measurements such as the Price-Jones, the methods mentioned are very crude.

The continuity of thought in reading the book is constantly interrupted by the repeated insertions of parenthetical remarks or words. The book is also heavily burdened by excessive terminology. There are several sentences that have been rendered meaningless by the translation and there is a constant interchange in the use of the word "signs" and "symptoms" disregarding correct connotation.

The material contained in the book bears valuable lessons to the practitioner who wishes to familiarize himself with hematologic text and it is a pity that a better organization of the available material was not created.